A PROSPECTIVE STUDY TO SEE THE IMPACT OF BARIATRIC SURGERY ON NON ALCOHOLIC FATTY LIVER DISEASE (NAFLD) IN MORBIDLY OBESE PATIENT

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BY

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DECLARATION

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Dedicated

to

my family

&

Teachers

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DR. Nikhil

ABBREVIATIONS

ALP : alkaline phosphatase

ALT : alanine aminotransferase

AST :aspartate aminotransferase

BMI : Body Mass Index

DAMPs : Damage-associated molecular patterns

FFA :free fatty acids

GLP-1 : Glucagon Like Peptide-1

LAGB : Laparoscopic adjustable gastric banding

NAFLD : Non Alcoholic Fatty Liver Disease

NASH : Non Alcoholic Steatohepatitis

PAMPs : pathogen-associated molecular patterns

RYGB :Roux-en-Y gastric bypass

SG :Sleeve gastrectomy

SGOT : serum glutamic-oxaloacetic transaminase

SGPT : serum glutamic-pyruvic transaminase

TG : Triglyceride

ABSTRACT

Introduction: Nonalcoholic fatty liver disease (NAFLD) affects over 80% of obese patients and is associated with the metabolic syndrome. NAFLD can progress into cirrhosis, which is associated to hepatocellular carcinoma and a liver transplant could be indicated. It represents the third cause of liver transplant, and it is expected to be first by 2025.Weight loss is strongly advocated as a central treatment for NAFLD, bariatric surgery is considered unique to obtain durable and permanent weight loss in morbid obesity and could reverse metabolic syndrome consequences as well as fatty liver disease.

Aim of Study: To evaluatethe effects of bariatripc surgery on non alcoholic fatty liver disease in morbidly obese patients

Materials & Methods: This prospective was conducted at Dayanand Medical College and Hospital, Ludhiana included patients admitted for morbid obesity with nonalcoholic fatty liver disease within 1.5 years of time period.

Result: There was a significant improvement in Fibrosis grade measured using fibroscan, Patients lying in F0-F1 group were 9/24 and in F3-F4 group were 11/24 preoperatively which was improved to 17/24 in F0-F1 group and 1/24 in F3-F4 group postoperatively at 6 months interval. Similarly NAFLD Fibrosis score showed significant improvement, Patients lying in F0-F2 group were 5/24 at the time of admission that was improved to 12/24 and 18/24 at 3 and 6 months interval respectively.

Conclusion: Bariatric surgery showed significant improvement in reversing the fibrosis grade and thus changing the NAFLD status of the morbidly obese patients.

Keywords: Bariatric surgery, weight loss, fatty liver, NAFLD, Metabolic surgery, NAFLD fibrosis score, fibroscan.

TABLE OF CONTENTS

S. NO.	TOPIC	PAGES
I	INTRODUCTION	1-3
II	AIMS AND OBJECTIVES	4
III	REVIEW OF LITERATURE	5-14
IV	MATERIAL AND METHODS	15-18
V	OBSERVATIONS AND RESULTS	19-31
VI	DISCUSSION	32-35
VII	SUMMARY AND CONCLUSION	36
VIII	BIBLIOGRAPHY APPENDICES – I - PROFORMA - MASTER CHART	37-45
	APPENDICES – II - APPROVAL LETTER OF THESIS PLAN - THESIS PLAN	

LIST OF TABLES

Table No.	Title	Page No.
1	FIBROSIS GRADE AND ITS INTERPRETATION USING FIBROSCAN	16
2	GENERAL RANGE OF LIVER FAT (CAP) SCORES AND THE MATCHING STEATOSIS GRADE	16
3	NAFLD FIBROSIS SCORE AND ITS CORRELATED SEVERITY	17
4	AGE WISE DISTRIBUTION OF PATIENTS	19
5	PATIENT GENDER	20
6	MEAN WEIGHT	21
7	CHANGE IN MEAN BMI POST SURGERY	22
8	HbA1c VALUE AT PRE-OPERATIVE AND POST-OPERATIVELY	24
9	MEAN SGOT VALUES OF THE PATIENTS AT TIME INTERVALS	25
10	MEAN SGPT VALUES OF THE PATIENTS AT TIME INTERVALS	26
11	MEAN ALBUMIN LEVELS OF THE PATIENTS	27
12	FIBROSIS GRADE USING FIBROSCAN AT ADMISSION	28
13	FIBROSIS GRADE USING FIBROSCAN AT 6 MONTHS INTERVAL	29
14	MEAN CAP VALUE OF PATIENT PRE AND POST OPERATIVELY AFTER BARIATRIC SURGERY	30
15	NAFLD FIBROSIS SCORE SEVERITY AND NUMBER OF PATIENTS	30

16	NAFLD FIBROSIS SCORE PRE AND POST-OPERATIVELY AFTER BARIATRIC SURGERY	31

LIST OF FIGURES

Fig. No.	Title	Page No.
1	AGE WISE DISTRIBUTION OF PATIENTS	19
2	GRAPHIC REPRESENTATION OF MALE TO FEMALE PATIENT IN PERCENTAGE	20
3	PRE & POST OPERATIVE WEIGHT OF PATIENTS AFTER BARIATRIC SURGERY	21
4	GRAPHIC REPRESENTATION OF PRE &POST OPERATIVE BMI OF PATIENTS AFTER BARIATRIC SURGERY	22
5	HISTORY OF DM PRE-OPERATIVELY	23
6	DIAGRAMMATIC REPRESENTATION OF MEAN HbA1c LEVEL PRE-OPERATIVELY AND POST-OPERATIVELY	24
7	DIAGRAMMATIC REPRESENTATION OF MEAN SGOT VALUES	25
8	DIAGRAMMATIC REPRESENTATION OF MEAN SGOT VALUES	26
9	DIAGRAMMATIC REPRESENTATION OF MEAN ALBUMIN LEVELS	27
10	FIBROSIS GRADING USING FIBROSCAN PRE-OPERATIVELY	28
11	FIBROSIS GRADING USING FIBROSCAN AT 6 MONTHS POST-OPERATIVELY	29
12	DIAGRAMMATIC REPRESENTATION OF MEAN NAFLD FIBROSIS SCORE	31

INTRODUCTION

Morbid obesity is strongly associated with nonalcoholic fatty liver disease (NAFLD), which is one of the most common causes of chronic liver disease worldwide. The current best treatment of NAFLD and NASH is weight reduction through life style modifications, antiobesity medication, and bariatric surgery. Importantly, bariatric surgery is the best alternative option for weight reduction if lifestyle modifications and pharmacological therapy have not yielded long-term success. Bariatric surgery is an effective treatment option for individuals who are grossly obese and associated with marked decrease in obesity-related morbidity and mortality. The most common performed bariatric surgery is Roux-en-Y gastric bypass (RYGB). The current evidence suggests that bariatric surgery in these patients will decrease the grade of steatosis, hepatic inflammation, and fibrosis. NAFLD *per se* is not an indication for bariatric surgery. Further research is urgently needed to determine (i) the benefit of bariatric surgery in NAFLD patients at high risk of developing liver cirrhosis (ii) the role of bariatric surgery in modulation of complications of NAFLD like diabetes and cardiovascular disease. The outcomes of the future research will determine whether bariatric surgery will be one of the recommended choices for treatment of the most progressive type of NAFLD.

Nonalcoholic fatty liver disease (NAFLD) is an increasingly recognized condition that occurs in all age groups and ethnicities.[1] Although first recognized in the 1930s , acknowledged clinically in the 1950s , and characterized histopathologically in the 1980s, only recently has the growing burden of disease gained notoriety.[2] Over this time, an assortment of terms has been used to represent this curiosity, including fatty liver hepatitis, nonalcoholic Laennec's disease, diabetes hepatitis, alcohol-like liver disease, steatonecrosis, and nonalcoholic steatohepatitis.[3] NAFLD has become the term of choice and describes a broad spectrum of hepatic conditions, which vary histologically. At one end of the spectrum lies simple steatosis, a relatively benign condition [4], in which there is an accumulation of lipids within the

hepatocytes >5% of liver weight.[1] At the other end is nonalcoholic steatohepatitis (NASH), which is steatosis in the presence of necroinflammatory hepatocellular changes [5]; this subset of patients is at increased risk of further progressing to fibrosis [1], cirrhosis, hepatocellular carcinoma and terminal liver failure.[6,7]

The diagnosis of NAFLD is often made fortuitously; steatosis and NASH are clinically indistinguishable [8] and in the absence of hepatic decompensation, largely asymptomatic.[3] Some patients may complain of malaise or right upper quadrant pain, with hepatomegaly being the only clinical observation.[4] Those at the malign end of the NAFLD spectrum may demonstrate evidence of cirrhosis, bruising, varices, ascites, splenomegaly, jaundice, and encephalopathy.[9] Identification of patients before they reach this point is therefore important, to avoid liver-related morbidity and mortality. A high index of clinical suspicion is needed to diagnose NAFLD, particularly in those whose serum liver function tests reveal an elevated ALT.[10] Meticulous history taking and investigation is also needed to exclude excess alcoholism (>10 g ethanol per day [2]) and other causes of liver disease or steatosis, viral, nutritional, drug/environmental/toxin induced, metabolic, genetically inherited, bowel diseases, and endocrine.[3]

Bariatric surgery has been shown to be superior to conservative measures with respect to weight reduction in the obese.[11] It can lower the long-term morbidity of obesity by up to 40% and alleviate the myriad of illnesses associated with it.[12] Since obesity is the key cause of NAFLD, this paper will look at bariatric surgery as a means of treatment for NAFLD. Worldwide, the number of surgical bariatric procedures has risen by an estimated 76% over the past ten years.[13] Procedures can be divided into 3 broad categories based on their mechanism of action [14]:

1. Restrictive procedures, which aim to restrict the amount of food that can be eaten by surgically reducing the size of the stomach. The restrictive procedures most commonly performed are vertical banded gastroplasty, laparoscopic adjustable gastric banding, and sleeve gastrectomy;

2. Malabsorptive procedures are less popular than restrictive procedures as they are more technically demanding to be performed and patients often develop nutritional deficiencies. Procedures aim to bypass a segment of the small bowel so that less food is absorbed (biliopancreatic diversion, biliopancreatic diversion with duodenal pouch);
3. Hybrid procedure aims to restrict food intake by creating a small gastric pouch which also limits absorption by bypassing the proximal small bowel—the Roux-en-Y gastric bypass (RYGB). It leaves 95% of the small bowel intact and so avoids many of the unwanted malabsorptive side effects such as diarrhoea and nutritional deficiencies. This is the most common surgical bariatric procedure performed.

   In this descriptive study, we have prospectively followed 24 cases (from January 2019 to June 2020) who presented to DMCH with morbid obesity with Nonalcoholic fatty liver disease, who underwent bariatric surgery as per standard National Institute of Health guidelines (BMI >35 kg/m2 or patients with BMI >32kg/m2 and associated comorbidities such as type-II diabetes mellitus, hypertension, obstructive sleep apnea and GERD) were included. Patients were analysed on the basis of pre-op and post op fibroscan and NAFLD fibrosis score. Fibroscan and NAFLD fibrosis score were compared. The presence of metabolic syndrome, anthropometric and biochemical variables were also assessed at baseline and at the time of second evaluation.

   Bariatric surgery in obese patients with NAFLD results in sustained and significant weight loss and improves NASH. Sleeve gastrectomy is considered safe and effective option for these patients, as long as appropriate long term follow up is provided.

AIMS AND OBJECTIVES

To evaluate the effects of bariatric surgery on non-alcoholic fatty liver disease in morbidly obese patients.

REVIEW OF LITERATURE

Non-alcoholic fatty liver disease (NAFLD) is increasingly recognized as a significant liver disease, and it covers the disease spectrum from simple steatosis with a risk of development of non-alcoholic steatohepatitis (NASH) to fibrosis, subsequent cirrhosis, end-stage liver failure, and liver cancer with a potential need for liver transplantation. NAFLD and NASH are closely related to obesity, metabolic syndrome, and type 2 diabetes (T2D). The role of gut hormones, especially glucagon-like peptide 1 (GLP-1), is important in NAFLD. Bariatric surgery has the potential for inducing great weight loss and may improve the symptoms of metabolic syndrome and T2D. Recent data demonstrated significant effects of bariatric surgery on GLP-1 and other gut hormones and important lipid metabolic and inflammatory abnormalities in the pathophysiology of NAFLD. Therefore, bariatric surgery may reverse the pathological liver changes in NAFLD and NASH patients. In the present review, NAFLD and NASH pathophysiology and the primary effects of bariatric surgery on metabolic pathways are described. A systematic review of the beneficial and harmful effects and focused on changes in liver disease severity in NAFLD and NASH patients following bariatric surgery is done.

NAFLD is characterized by lipid deposition in hepatocytes with progressive hepatocellular degeneration, inflammation, and fibrotic parenchymal changes. It may result in progressive liver injury and is associated with increased mortality from cardiovascular disease, malignancy, and liver failure. Morbid obesity and metabolic syndrome are also strongly linked with NAFLD and co-occurring health issues likely related to obesity.

Alarming increases in obesity, coupled with changes towards unhealthy lifestyles and dietary habits have contributed to a dramatic increase in non-alcoholic fatty liver disease (NAFLD), which affects 25%-30% of the general population.[1] NAFLD is most often asymptomatic and consists of a disease spectrum ranging from simple steatosis (NAFL)

and steatohepatitis (NASH) to fibrosis and cirrhosis, with significant clinical consequences, including but not limited to ascites, varices, hepatic encephalopathy, liver cancer and liver transplantation or early death.[6] NASH may develop with hepatic inflammation, hepatocellular injury, macrophage and hepatic stellate cell activation in patients with simple steatosis. Non-alcoholic fatty liver disease is the most common cause of elevated liver enzymes. Within the NAFLD spectrum, only NASH progresses to cirrhosis and hepatocellular carcinoma. With the growing epidemic of obesity, the prevalence and impact of NAFLD continues to increase, making NASH potentially the most common cause of advanced liver disease in coming decades. If untreated, NASH may progress to cirrhosis. NASH-induced cirrhosis is fast becoming the most common indication for liver transplantation, which is strongly associated with poor quality of life.[5]The prevalence of major causes of CLD remained stable, except for NAFLD, which increased steadily, along with the prevalence of metabolic conditions. Given the increasing rates of obesity, NAFLD prevalence is expected to contribute substantially to the burden of CLD.[15]

IS NON-ALCOHOLIC FATTY LIVER: ANOTHER FEATURE OF THE METABOLIC SYNDROME?

There is a strong association between non-alcoholic fatty liver and features of the metabolic syndrome, suggesting a simultaneous insulin resistance and decreased sensitivity to leptin.[2] The association between NAFLD and type 2 diabetes mellitus (T2DM) has been well established, which could be explained by the insulin-resistance and compensatory hyperinsulinemia progressing to defective lipid metabolism and hepatic triglyceride (TG) accumulation in NAFLD or to β-cell dysfunction in T2DM.[9]Peripheral insulin resistance, increased fatty acid beta oxidation, and hepatic oxidative stress are present in both fatty liver and NASH, but NASH alone is associated with mitochondrial structural defects.[16]

BARIATRIC SURGERY AND OBESITY

Bariatric surgery is the most effective treatment for obesity and also greatly improves glycaemic control, often within days after surgery,

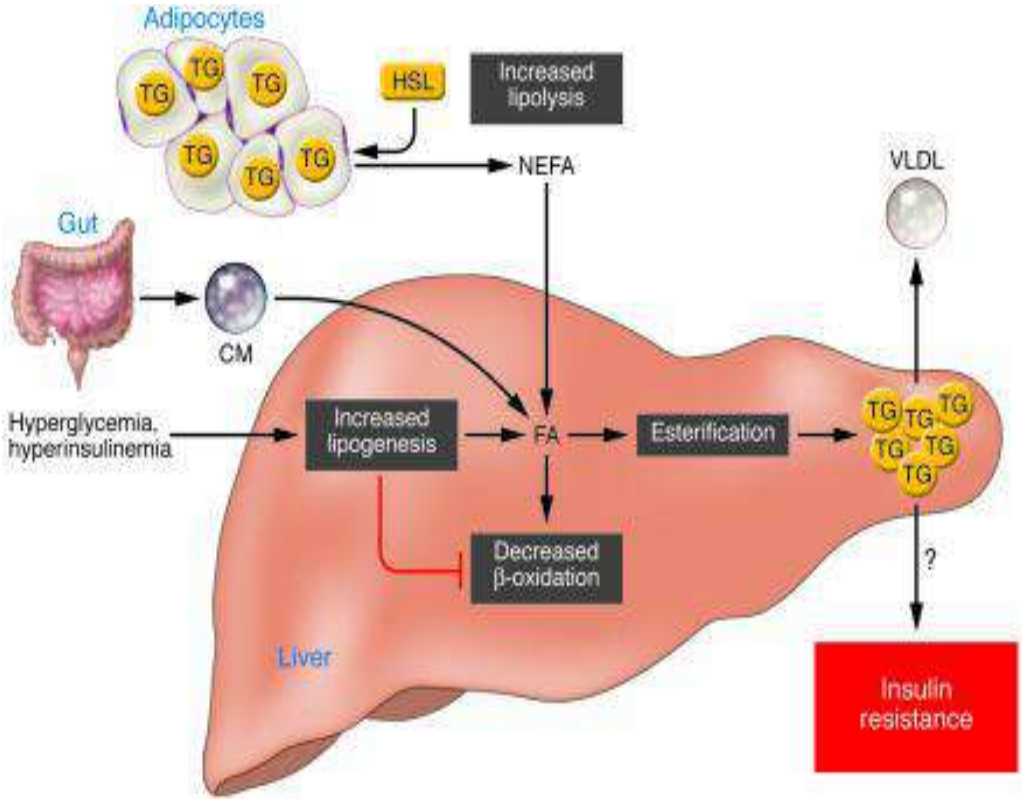
independently of weight loss. Laparoscopic adjustable gastric banding (LAGB) was designed as a purely restrictive procedure, whereas vertical sleeve gastrectomy (VSG) and Roux-en-Y gastric bypass (RYGB) induce changes in appetite through regulation of gut hormones, resulting in decreased hunger and increased satiation. Thus, VSG and RYBG more frequently result in remission of type 2 diabetes than does LAGB. With all three of these procedures, remission of diabetes is associated with early increases in insulin sensitivity in the liver and later in peripheral tissues; VSG and RYBG are also associated with improved insulin secretion and an exaggerated postprandial rise in glucagon-like peptide 1. The vagal pathway could have a role in the neurohumoral regulatory pathways that control appetite and glucose metabolism after bariatric surgery. Recent research suggests that changes in bile acid concentrations in the blood and altered intestinal microbiota might contribute to metabolic changes after surgery, but the mechanisms are unclear. Elucidation of these mechanisms is providing knowledge about bodyweight regulation and the pathophysiology of type 2 diabetes, and could help to identify new drug targets and improved surgical techniques.Bariatric surgery is an efficient treatment of obesity as it causes sustained weight loss with potential reductions in hepatic fat, inflammation and fibrosis.[8,17] Roux-en-Y gastric bypass surgery (RYGB) is the most effective treatment for obesity.[18] This procedure improves glycaemic control, and T2D patients experience a reduced need for antidiabetic medication within days after surgery.[17] Sleeve gastrectomy (SG) and adjustable gastric banding (AGB) are alternative surgical approaches that significantly reduce gastric volume without changing the upper gastrointestinal tract anatomy. SG has gained popularity in recent years and been established as a comparable method to RYGB. In contrast, AGB is associated with less weight loss than RYGB surgery. The indication for bariatric surgery is severe obesity with or without T2D and/or other comorbidities.[17] Bariatric surgery induced the disappearance of NASH from nearly 85% of patients and reduced the pathologic features of the disease after 1 year of follow-up. It could be a therapeutic option for

appropriate morbidly obese patients with NASH who do not respond to lifestyle modifications.[8]Maciejewski ML and et al, in their study evaluated, that How much weight loss can be expected on average 10 years after undergoing Roux-en-Y gastric bypass (RYGB) and how does weight loss 4 years after bariatric surgery compare among patients undergoing RYGB, adjustable gastric banding, and sleeve gastrectomy? **Findings** were, patients undergoing RYGB lost 21% more of their baseline weight than matched nonsurgical patients at 10 years, and RYGB induced significantly greater weight loss than sleeve gastrectomy or adjustable gastric banding at 4 years.[18]

PATHOGENESIS OF NAFLD

The pathogenic mechanisms for the development and progression of NAFLD are complex and multifactorial.[19]Insulin resistance (IR) and mitochondrial dysfunction play a central role in the pathophysiology of nonalcoholic fatty liver disease (NAFLD). Genetic factors and epigenetic modifications occurring in the liver contribute to the IR phenotype.[20]Genetic and epigenetic factors affect the development of NAFLD and NASH progression and potentially influence or modify risk factors.[20-23]An allele in *PNPLA3*, was strongly associated with increased hepatic fat levels and with hepatic inflammation. The allele was most common in Hispanics, the group most susceptible to NAFLD; hepatic fat content was > 2-fold higher in *PNPLA3* homozygotes than in non-carriers.[21]The TM6SF2 variant encoding p.Glu167Lys was also associated with higher circulating levels of alanine transaminase, a marker of liver injury, and with lower levels of low-density lipoprotein-cholesterol (LDL-C), triglycerides and alkaline phosphatase.[22]Dietary sugars, fat, adipose tissue lipolysis, and de novo lipogenesis contribute to increased hepatic fat influx and accumulation in obese patients. Nonalcoholic fatty liver disease (NAFLD) is characterized by the accumulation of excess liver triacylglycerol (TAG), inflammation, and liver damage.[24] Obese patients exhibit increased adipose tissue mass, which leads to adipocyte dysfunction, including insulin resistance, increased lipolysis and apoptosis, and results in local inflammation and cytokine release. Insulin resistance reduces insulin-induced inhibition

of lipolysis, and negatively affects the ability of the adipose tissue to store fat, which results in increased free fatty acids in the blood. Insulin resistance induces further insulin secretion, which instigates high blood insulin levels.[25,26]Abnormalities of triglyceride storage and lipolysis in insulin-sensitive tissues are an early manifestation of conditions characterized by insulin resistance and are detectable before the development of postprandial or fasting hyperglycaemia. Increased free fatty acid (FFA) flux from adipose tissue to non-adipose tissue, resulting from abnormalities of fat metabolism, participates in and amplifies many of the fundamental metabolic derangements that are characteristic of the insulin resistance syndrome and type 2 diabetes. It is also likely to play an important role in the progression from normal glucose tolerance to fasting hyperglycaemia and conversion to frank type 2 diabetes in insulin resistant individuals. Adverse metabolic consequences of increased FFA flux, to be discussed in this review, are extremely wide ranging and include, but are not limited to: 1) dyslipidaemia and hepatic steatosis, 2) impaired glucose metabolism and insulin sensitivity in muscle and liver, 3) diminished insulin clearance, aggravating peripheral tissue hyperinsulinemia, and 4) impaired pancreatic beta-cell function.[25] The development of NAFLD and NASH are closely linked to an excess flow of free fatty acids (FFA) arising from dysfunctional/insulin resistant adipose tissue causing ectopic fat deposition in many organs. In the liver, when chronic lipid supply surpasses the metabolic ability to adapt it will induce hepatocellular damage as FFA are redirected into harmful pathways of non-oxidative metabolism with intracellular accumulation of toxic lipid-derived metabolites.[26]



Metabolic defects leading to the development of hepatic steatosis**.** Different sources of fatty acids contribute to the development of fatty liver. Under conditions of insulin resistance, insulin does not adequately inhibit HSL, and lipolysis in white adipose tissue is not suppressed. Therefore, peripheral fats stored in adipose tissue flow to the liver by way of plasma NEFAs.Dietary fatty acids are also taken up by the liver through the uptake of intestinally derived chylomicron (CM). In addition, the combination of elevated plasma glucose (hyperglycaemia) and insulin concentrations (hyperinsulinemia) promotes de novo fatty acid synthesis (lipogenesis) and impairs β-oxidation, thereby contributing to the development of hepatic steatosis. After the esterification step (conversion of FAs into TGs) TG can then be stored as lipid droplets within hepatocytes or secreted into the blood as VLDL.[27]

RETHINK THE CAUSES OF NASH!

A significant body of evidence now forces us to rethink the causes of NASH. Once thought to be a disease caused by triglyceride accumulation in hepatocytes with subsequent oxidant stress and lipid peroxidation causing inflammation and fibrosis, new data from animal

studies and a limited number of human studies now provide convincing evidence that triglyceride accumulation does not cause insulin resistance or cellular injury in the liver. The lipotoxic liver injury hypothesis for the pathogenesis of NASH suggests that we need to focus our therapeutic efforts on reducing the burden of fatty acids going to the liver or being synthesized in the liver. This can be accomplished by improving insulin sensitivity at the level of adipose tissue to prevent inappropriate peripheral lipolysis and by preventing unnecessary de novo lipogenesis in the liver. Excess carbohydrates are the major substrates for de novo lipogenesis, and thus, reducing carbohydrate consumption through dietary changes and increasing muscle glucose uptake through exercise remain important cornerstones of treatment and prevention of lipotoxic liver injury, a disease hitherto called NASH.[28] Fatty acids, DAMPs and pathogen-associated molecular patterns (PAMPs), e.g., bacteria and endotoxins, likely originating from a leaky gut, are the primary inducers of hepatic inflammation, which involves activation of resident and recruited macrophages in the liver.[29,30] Macrophage activation results in pro-inflammatory cytokine secretion and the activation of hepatic stellate cells into myofibroblasts, which secrete the collagen that contributes to extracellular matrix formation. Myofibroblasts are also directly responsive to cytokines, DAMPs and PAMPs, thus further propagating fibrosis formation.[31]

METABOLIC EFFECTS OF BARIATRIC SURGERY

Bariatric surgery has tremendous effects on metabolic functions. Buchwald et al[32] performed a meta-analysis of 136 studies that assessed the impact of bariatric surgery on metabolic outcomes and reported a complete resolution of T2D in more than 75% of diabetic patients and an excessive weight loss of almost 60%. A review of key results from the Swedish Obese Subjects study reported a 72% remission rate of T2D two years' post-bariatric surgery.[31] Several other studies demonstrated that RYGB and SG were superior to conventional pharmacological therapy in achieving glycaemic control in T2D patients.[33-36] In a study by Ackerman and et al, they did study on Observations on the improvements in carbohydrate metabolism in

diabetic and other morbidly obese patients after jejunoileal bypass. In their study, In most morbidly obese patients with diabetes, fasting plasma glucose levels decreased immediately after jejunoileal bypass operations, with patients often becoming normoglycemic before discharge from the hospital. All 12 patients who had required insulin or orally administered hypoglycaemic agents preoperatively were able to discontinue the medication shortly after the operation. Oral glucosetolerance test curves in all morbidly obese patients had a flattened pattern, in the normal range, postoperatively. Serum insulin levels, which had been elevated preoperatively, decreased significantly, both in fasting and post glucose determinations.[37]

NAFLD is closely associated with obesity and T2D, and the mechanisms implicated in improving obesity and T2D following bariatric surgery likely play important roles in the resolution of NAFLD. Several mechanisms independent of weight loss are involved in the initial metabolic responses to RYGB and SG procedures and maintaining these improvements over the long term, despite the obvious causality between weight loss and improvements in T2D and NAFLD.

The almost immediate metabolic benefits of bariatric surgery, independent of any significant weight loss, have been known for decades[35] but are striking nonetheless.[30,36,37]Roux-en-Y gastric bypass (RYGB) improves glycaemic control within days after surgery, and changes in insulin sensitivity and β-cell function are likely to be involved.[38]Three primary mechanisms are involved in the improved glycaemic control associated with the RYGB and SG procedures: (1) early improved hepatic insulin sensitivity due to the post-surgery calorie restriction; (2) late improved peripheral insulin sensitivity due to weight loss; and (3) improved post-prandial insulin secretion due to a rise in glucagon-like peptide 1 (GLP-1) secretion.Roux-en-Y gastric bypass (RYGB) surgery induces weight loss of 20-30% that is maintained for 20 years. In patients with type 2 diabetes, the glucose-lowering effect of RYGB is superior to conventional anti-diabetic therapy and often occurs within days after surgery.[13] β-Cell function improves in patients

with type 2 diabetes in response to an oral glucose stimulus after Roux-en-Y gastric bypass (RYGB) surgery. This has been linked to the exaggerated secretion of glucagon-like peptide 1 (GLP-1), but causality has not been established.[39] Several studies investigated the post-surgical metabolic changes, and whether the change in the release of gut hormones or surgery-induced restriction of food intake provides the essential effects on glycaemic control remains controversial. Jørgensen et al[13,39] found an increase in post-prandial GLP-1, insulin secretion and hepatic insulin sensitivity within days after RYGB, and this increase was sustained for at least 1 year in diabetic and non-diabetic-matched subjects, which is consistent with other RYGB and SG studies.[38,40,41] The accelerated transport of nutrients into more distal parts of the small intestine may further explain the exaggerated GLP-1 response because a higher density of L cells are found in this area.[38] Notably, GLP-1 may have beneficial gene-regulatory effects on fatty acid oxidation and insulin sensitivity in hepatocytes[42,43], but these findings require confirmation in humans. Postprandial glucagon responses also increase post-operatively[13,44] despite the inhibitory effects of GLP-1 on glucagon secretion.[39] Calorie restriction is the leading mechanism of the early metabolic changes after bariatric surgery, but the gut hormones, particular GLP-1, remain crucial for the fine-tuning of the glycaemic control and post-prandial insulin secretion.[45]

A hyperinsulinemic clamp study of Bojsen-Møller et al[38] demonstrated that peripheral insulin sensitivity, as assessed using glucose disposal and suppression of fatty acids, increased after three months in relation to the surgery-induced weight loss. Lifestyle modifications exert the same powerful effects on glycaemic control and NAFLD[44,46], but this intervention generally fails to sustain the short-term weight loss.[33] Notably, bariatric surgery is superior in maintaining calorie restriction and long-term weight loss, which is facilitated at least partially by the reduced appetite observed post-operatively.[47,48] The long-term reduced appetite may be attributable to a favourable shift in the anorectic gut hormones GLP-1 and peptide-YY (PYY).[43,49] An

antagonist study using exendin9–39 and a dipeptidyl peptidase 4 (DPP-4) inhibitor (blocking the DPP-4-mediated formation of active PYY from its precursor) in RYGB-operated subjects demonstrated a 20% increase in food intake.[50]

HEPATIC EFFECTS OF BARIATRIC SURGERY

Bariatric surgery affects NAFLD not only through a rapid and substantial weight loss but also via simultaneous effects on important lipid metabolic and inflammatory pathways involved in NAFLD pathophysiology.[51,52]Weight loss induced by GBP (Gastric bypass surgery) normalizes the metabolic abnormalities involved in the pathogenesis and pathophysiology of NAFLD and decreases the hepatic expression of factors involved in the progression of liver inflammation and fibrosis.[51] Due to the association between the quantity of adipose tissue and concentrations of interleukin-6 (IL-6) and tumor necrosis factor (TNF-α), work by Viana and et al, aimed to assess the effects of Roux-en-Y gastric bypass (RYGB) and sleeve gastrectomy (SG) procedures on serum IL-6 and TNF-α concentrations and concluded RYGB and SG procedures demonstrated a similar impact on adipokine levels in women 1 year post-surgery. Both techniques may improve the course of chronic diseases and the state of inflammation associated with obesity.[52]Bariatric surgery promotes changes in three crucial metabolic areas influencing NAFLD: improved glucose homeostasis, improved lipid metabolism and reduced inflammatory activity. These effects are followed by significant effects on liver abnormalities in NAFLD and NASH patients. A systematic review and meta-analysis[53] in 2008 assessed the histological effects of bariatric surgery in NAFLD patients. A Cochrane review in 2010 reported more discrete conclusions.[54] These authors were not able to identify any randomized clinical trials at that time and advised caution even though several reports demonstrated potential favourable effects of bariatric surgery. Most studies reported beneficial effects on steatosis, and more than half of the studies demonstrated significant improvements in histological inflammation

MATERIALS AND METHODS

This prospective study was conducted at Dayanand Medical College and Hospital, Ludhiana and includes patients admitted for morbid obesity with nonalcoholic fatty liver disease in a 1.5 years of time period.

In this prospective study, all consecutive patients undergoing bariatric surgery (between January 2019 and June 2020) as per standard National Institute of Health guidelines were included; that is, patients with body mass index (BMI)>35 kg/m2 or patients with BMI>32 kg/m2 and associated co-morbidities, such as type 2 diabetes, hypertension, obstructive sleep apnea, and gastroesophageal reflux disease.

•Patients were analysed on the basis of pre-op and post-op FIBROSCAN and NAFLD fibrosis score, fibroscan was done at 6 months of interval of the surgery and nafld fibrosis score was conducted at 3 ± 2 months of time period

SOURCE OF DATA

Patients coming in Department of Surgery of Dayanand Medical College and Hospital with morbid obesity.

METHOD OF COLLECTION OF DATA

Patients coming in Department of Surgery of Dayanand Medical College and Hospital with morbid obesity were taken up for the study. A group of morbidly obese patients with a body mass index > 35 kg/m2 who underwent sleeve gastrectomy and mini gastric bypass operations had paired fibroscan with nafld fibrosis score, the first at surgery and the second after 6 months of weight loss. Fibroscan and nafld fibrosis score were assessed and compared. The presence of metabolic syndrome, anthropometric and biochemical variables was also assessed at baseline and at the time of second evaluation.

FIBROSCAN

Fibroscan is a non-invasive device that assesses the ‘hardness’ (or stiffness) of the liver via the technique of transient elastography. Liver hardness is evaluated by measuring the velocity of a vibration wave (also called as a ‘shear wave’) generated on the skin. Shear wave

velocity is determined by measuring the time the vibration takes to travel to a partcular depth inside the liver.

Because fibrosis tissue is harder than normal liver, the degree of hepatic fibrosis can be inferred from liver hardness. Fibroscan measures scarring by measuring the stiffness of the liver. The fibrosis result is measured in kilopascals (kPa). It's normally between 2 and 6 kPa. The highest possible result is 75 kPa.

TABLE 1: FIBROSIS GRADE AND ITS INTERPRETATION USING FIBROSCAN

Fibrosis grade	Interpretation
F0	No liver scarring
F1	Mild liver scarring
F2	Moderate liver scarring
F3	Severe liver scarring
F4	Advanced liver scarring

The Controlled Attenuation Parameter (CAP) measures the fat in the liver. This score is measured in decibels per meter (dB/m) and it ranges from 100 to 400 dB/m. Table 2. shows general range of liver fat (CAP) scores and the matching steatosis grade which suggests the amount of fatty change in the liver.

TABLE 2: GENERAL RANGE OF LIVER FAT (CAP) SCORES AND THE MATCHING STEATOSIS GRADE

CAP score	Steatosis grade	Amount of Liver showing fatty changes
150-238 dB/m	S0	0 to 10%
238-260 dB/m	S1	11 to 33%
260-290 dB/m	S2	34 to 66%
Higher than 290 dB/m	S3	67% or more

NAFLD Fibrosis Score:

NAFLD Fibrosis score was calculated using the online calculator, It estimates amount of scarring in the liver based on several laboratory tests. It is a non-invasive scoring system composed of routinely measured and easily available clinical and laboratory variables to discriminate between the presence or absence fibrosis in patients with NAFLD.

NAFLD Fibrosis score is calculated using Age, BMI, Impaired fasting flucose/diabetes status, and other laboratory tests – AST, ALT, Platelet count and Albumin levels.

TABLE 3: NAFLD FIBROSIS SCORE AND ITS CORRELATED SEVERITY

NAFLD score	Correlated Fibrosis Severity
< -1.455	F0 – F2
- 1.455 to 0.675	Indeterminate score
> 0.675	F3 – F4

Table 3. shows that in patients with NAFLD fibrosis score above 0.675, the presence of advanced liver fibrosis can be diagnosed with high accuracy. In patients with an NAFLD fibrosis score below -1.455 advanced liver fibrosis can be excluded with high accuracy. Scores between – 1.455 and 0.675 are considered indeterminate.

INCLUSION CRITERIA

Patients with body mass index (BMI)>35 kg/m2 or patients with BMI>32 kg/m2 and associated co-morbidities, such as type 2 diabetes, hypertension, obstructive sleep apnea, and gastroesophageal reflux disease.

EXCLUSION CRITERIA

1) Patients undergoing revision surgery, (2) patients with alcohol consumption>21 drinks/week for men and>14 drinks per week for women, (3) patients with hepatitis B or hepatitis C infection, (4) patients

on drugs known to induce fatty liver or insulin sensitization such as estrogens, tamoxifen, amiodarone, methotrexate, and glitazones.

NUMBER OF CASES

Patients admitted with above mentioned inclusion criteria and nonalcoholic fatty liver disease in 1.5 years of time period extending from January 2019 to June 2020.

STATISTICAL ANALYSIS:

Data were described in terms of range; mean ±standard deviation (± SD), frequencies (number of cases) and relative frequencies (percentages) as appropriate. Comparison of quantitative variables was done using Paired t-test and Wilcoxon rank test for independent samples for parametric and non-parametric data respectively. A probability value (p value) less than 0.05 was considered statistically significant. All statistical calculations were done using (Statistical Package for the Social Science)SPSS 21version (SPSS Inc., Chicago, IL, USA)statistical program for Microsoft Windows.

OBSERVATIONS AND RESULTS

The present study was conducted in Department of Surgery, DMCH Ludhiana, over a period of one and half year from September 2018 to May 2020. 24 morbidly obese patients underwent bariatric surgery were included in the study which were followed up for 6 months. Results of the study are presented below:

AGE:

Table 4 and figure 1 ,shows that out of 24 patients in our study, 10(41.7%) number of patients were age group more than 60, while 10(41.7%) belonged to age group below 40years and 4(16.6%) to the age group 41-50years , mean age (standard deviation) was 45±10 years.

TABLE 4: AGE WISE DISTRIBUTION OF PATIENTS

Age	No. of cases	Percentage
< 40	10	41.7%
41-50	4	16.6%
> 60	10	41.7%
Total	24	100.0%

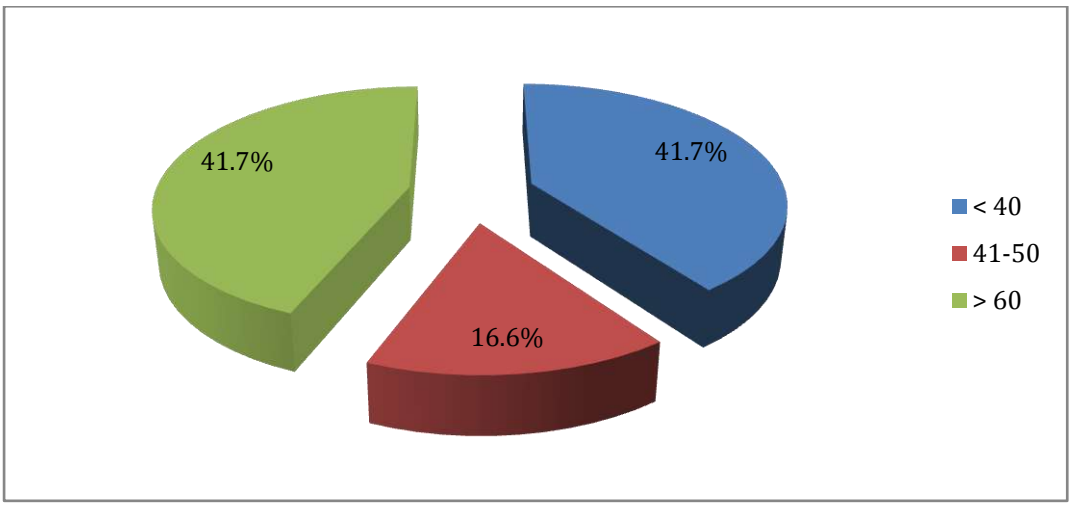


FIGURE 1: AGE WISE DISTRIBUTION OF PATIENTS

GENDER

Table 5 and Figure 2 shows that of the 24 patients, majority of our case were female 17(71%) of patients and 7(29%) patients were males

TABLE 5: PATIENT GENDER

Sex	No. of cases	Percentage
FEMALE	17	71.0%
MALE	7	29.0%
Total	24	100.0%

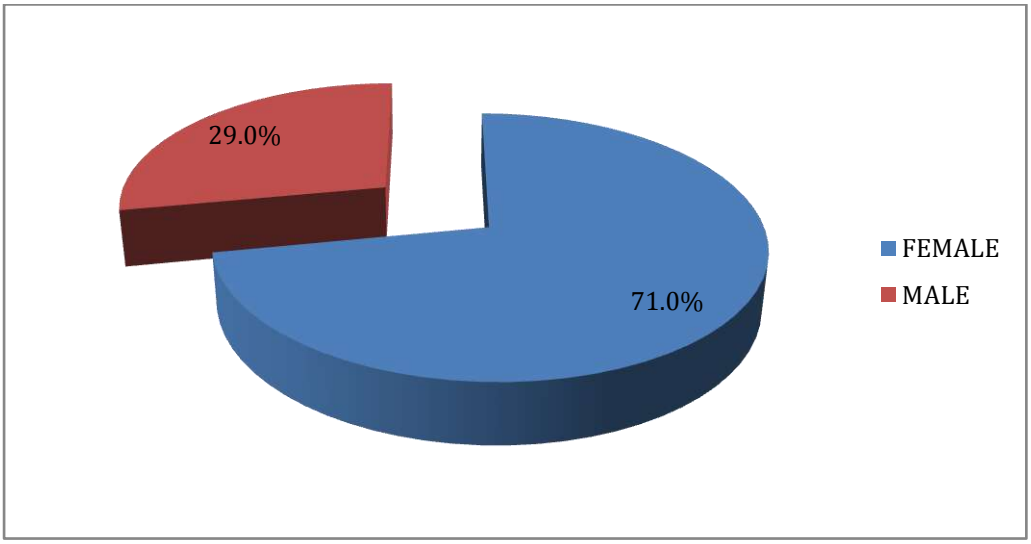


FIGURE 2: GRAPHIC REPRESENTATION OF MALE TO FEMALE PATIENT IN PERCENTAGE

WEIGHT

Table 6 and Figure 3. shows weight loss after the bariatric surgery . Weight was recorded before surgical intervention, which was 115.39±20.84. Post surgery at 3 month and 6 month follow-up demonstrated an overall decrease in mean weight to 96.15 (3months mark) to 87.25 (6months mark)respectively, with a significant difference statistically (p-value<0.05).

TABLE 6: MEAN WEIGHT

WEIGHT				Z	p-value	Difference	
	N	Mean	SD			Mean	SD
Pre operatively	24	115.39	20.84				
After 3 months	24	96.15	14.05	10.862	0.000	19.24	8.68
After 6 months	24	87.25	10.32	10.388	0.000	28.14	13.27

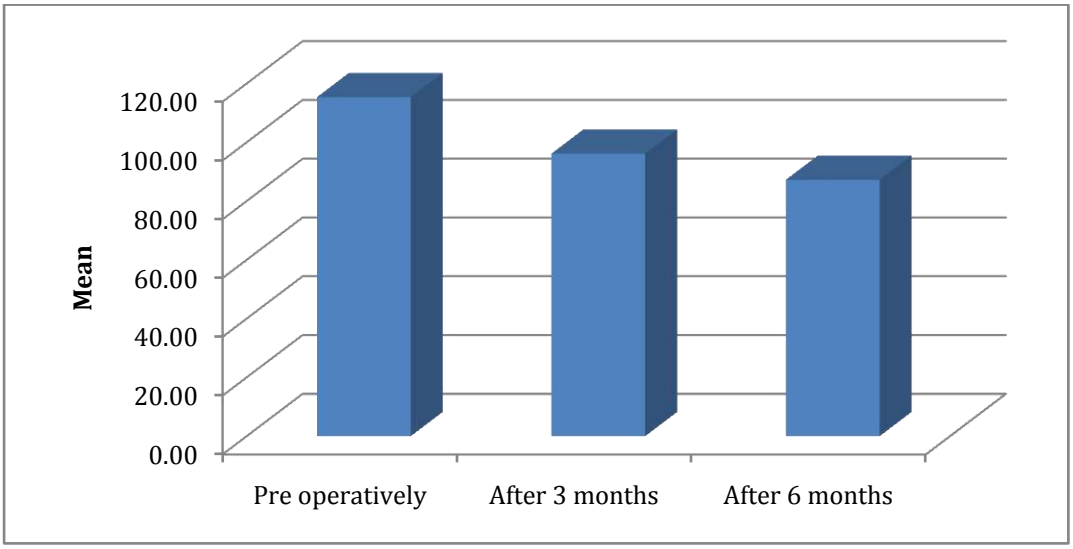


FIGURE 3: PRE & POST OPERATIVE WEIGHT OF PATIENTS AFTER BARIATRIC SURGERY

BODY MASS INDEX

Table 7 and Figure 4shows the BMI (Body Mass Index) as calculated by weight (in kg) divided by height (in m2). This is the main parameter depending upon which patient becomes eligible for bariatric surgery. BMI was recorded before surgical intervention which was 47.90±9.28, decrease in mean BMI from 48.10 to 39.98 (3-month mark) to 36.27(6-month mark) was seen at 3 and 6 months post-op time period respectively with a significant difference statistically (p-value<0.05).

TABLE 7: CHANGE IN MEAN BMI POST SURGERY

BMI				Z	p-value	Difference	
	N	Mean	SD			Mean	SD
Pre operatively	24	47.90	9.28				
After 3 months	24	39.98	6.18	9.929	0.000	7.92	3.91
After 6 months	24	36.27	4.36	9.721	0.000	11.64	5.86

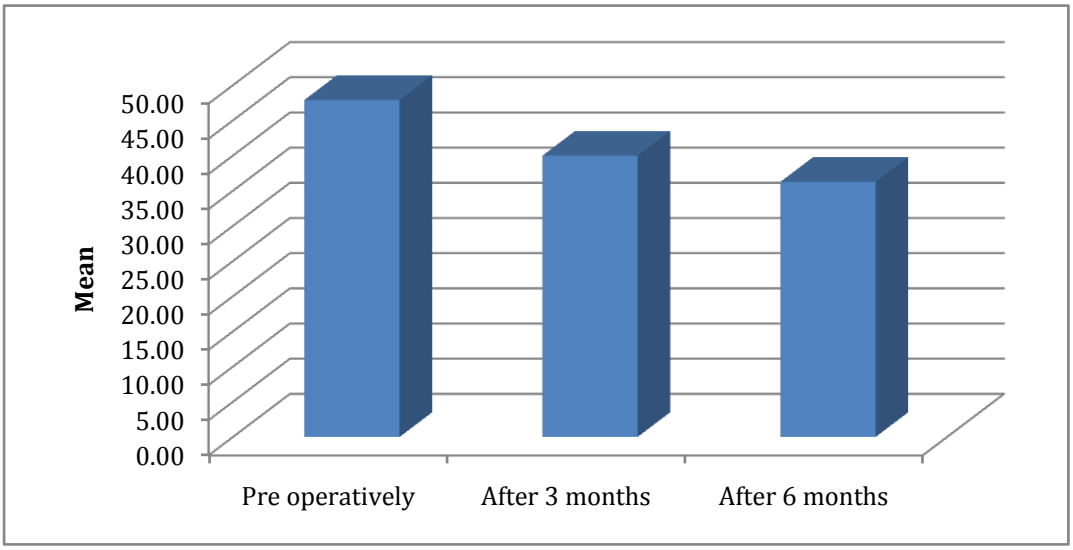


FIGURE 4: GRAPHIC REPRESENTATION OF PRE &POST OPERATIVE BMI OF PATIENTS AFTER BARIATRIC SURGERY

DIABETES MELLITUS

In our study, among the 24 patients 10 of them were diabetic and 14 of them were non-diabetic [figure5],

Table 8 and figure 6 shows DM status of the patient pre and post operatively, there was significant improvement in diabetic status of the patient with decrease in HbA1c level of patient pre-operatively and post-operatively at 3 and 6 months intervals.

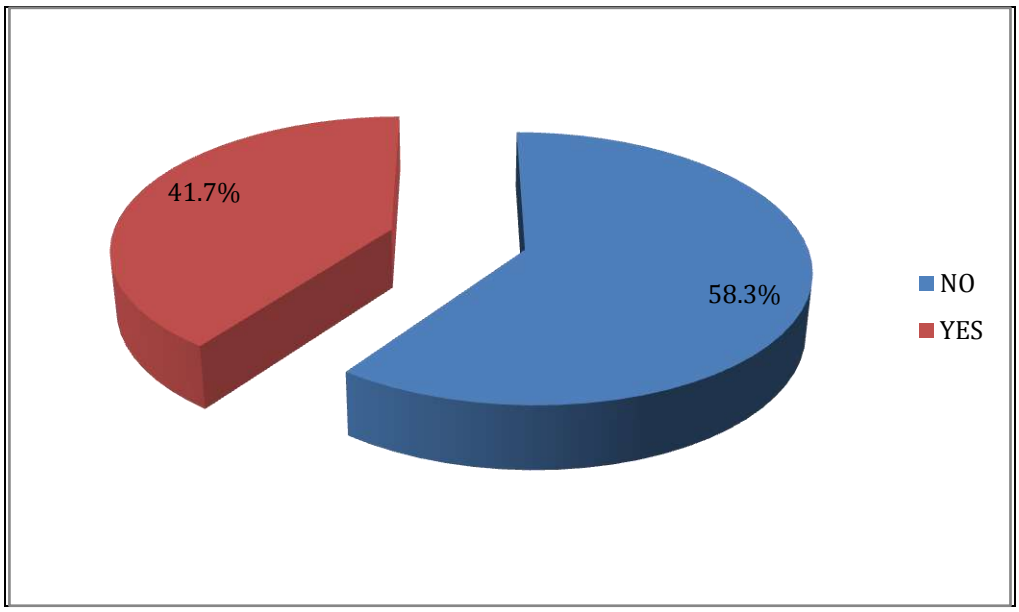


FIGURE 5: HISTORY OF DM PRE-OPERATIVELY

TABLE 8: HbA1c VALUE AT PRE-OPERATIVE AND POST-OPERATIVELY

HbA1c				t	p-value	Difference	
	N	Mean	SD			Mean	SD
Pre operatively	24	6.77	1.34				
After 3 months	24	6.27	0.96	4.749	0.000	0.50	0.52
After 6 months	24	5.74	0.57	5.339	0.000	1.03	0.94

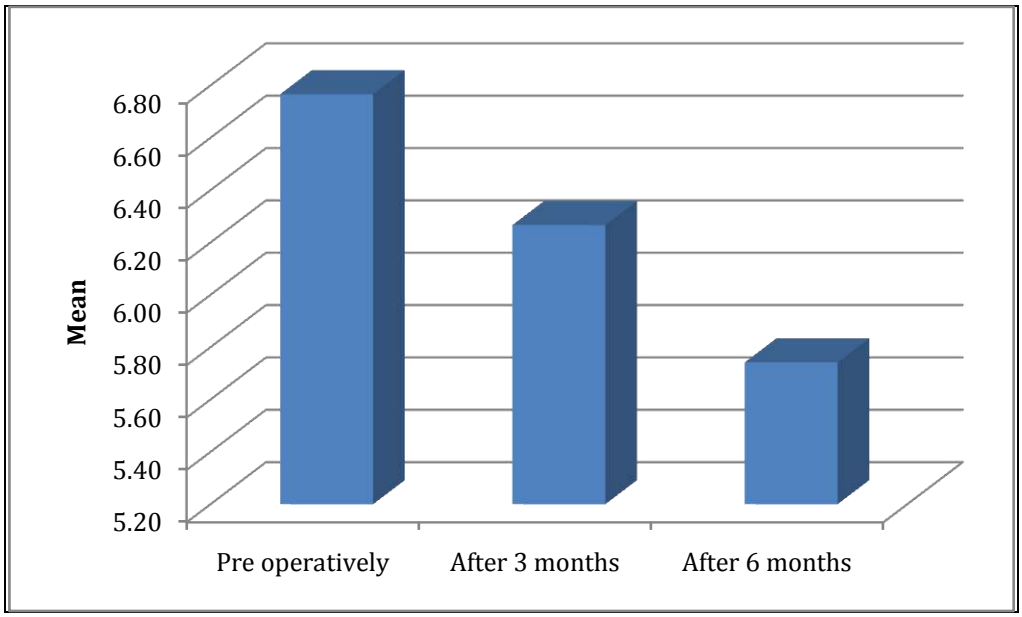


FIGURE 6: DIAGRAMMATIC REPRESENTATION OF MEAN HbA1c LEVEL PRE-OPERATIVELY AND POST-OPERATIVELY

SGOT / AST:

Table 9 and figure 7 shows SGOT status of the patient pre and post operatively, the mean SGOT was 29.33 at the time of admission, 27.63 at 3 months interval and 28.13 at 6 months interval. Which was insignificant change (p>0.05) .

TABLE 9: MEAN SGOT VALUES OF THE PATIENTS AT TIME INTERVALS

SGOT				t	p-value	Difference	
	N	Mean	SD			Mean	SD
Pre operatively	24	29.33	9.83				
After 3 months	24	27.63	5.50	1.163	0.257	1.71	7.20
After 6 months	24	28.13	5.45	0.772	0.448	1.21	7.67

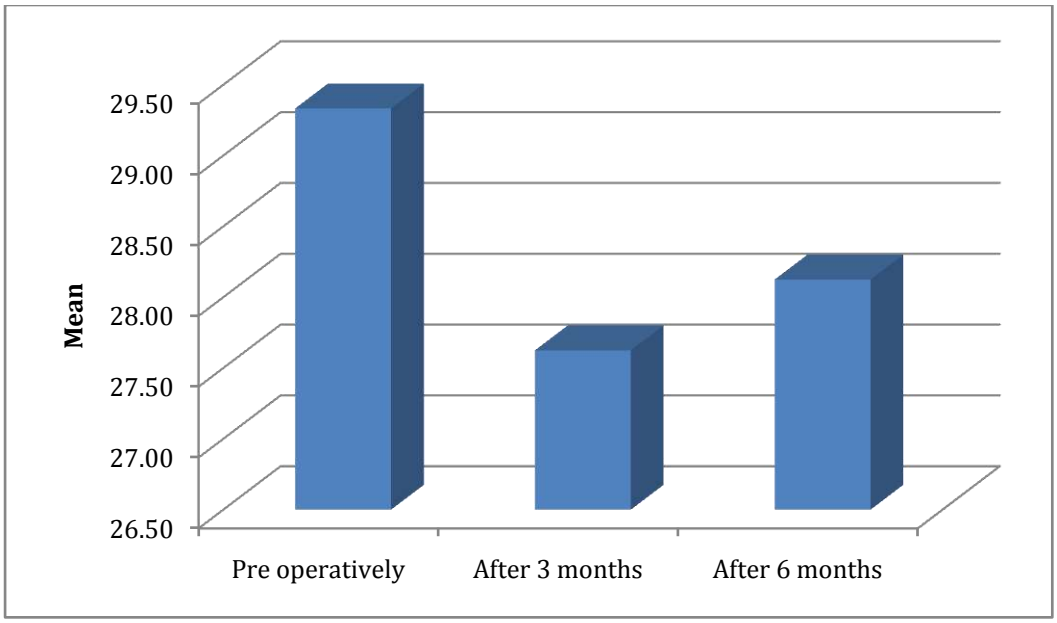


FIGURE 7: DIAGRAMMATIC REPRESENTATION OF MEAN SGOT VALUES

SGPT / ALT:

Table 10 and figure 8 shows SGPT status of the patient pre and post operatively, the mean SGOT was 33.38 at the time of admission, 29.21 at 3 months interval and 28.00 at 6 months interval. Which showed a significant change ($p<0.05$).

TABLE 10: MEAN SGPT VALUES OF THE PATIENTS AT TIME INTERVALS

SGPT				Z	p-value	Difference	
	N	Mean	SD			Mean	SD
Pre operatively	24	33.38	13.45				
After 3 months	24	29.21	6.23	2.513	0.019	4.17	8.12
After 6 months	24	28.00	8.14	2.663	0.014	5.38	9.89

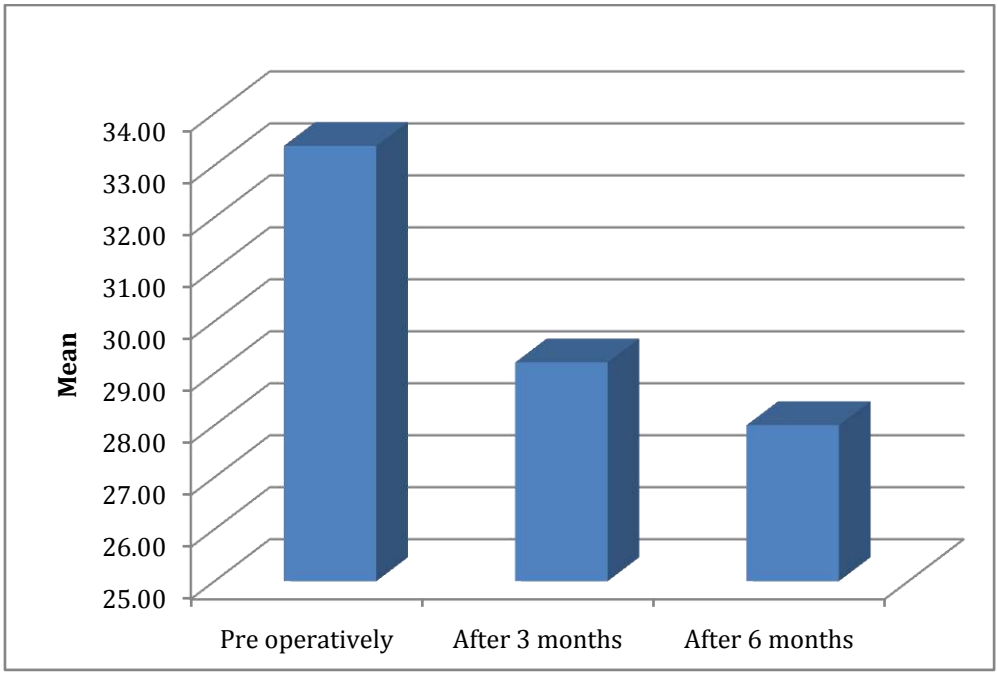


FIGURE 8: DIAGRAMMATIC REPRESENTATION OF MEAN SGOT VALUES

Albumin:

Table 11 and figure 9 shows Albumin status of the patient pre and post operatively, the mean albumin was 4.10 at the time of admission, 4.25 at 3 months interval and 4.64 at 6 months interval, which showed a significant change ($p<0.05$).

TABLE 11: MEAN ALBUMIN LEVELS OF THE PATIENTS

ALB				t	p-value	Difference	
	N	Mean	SD			Mean	SD
Pre operatively	24	4.10	0.43				
After 3 months	24	4.25	0.33	-2.136	0.044	-0.15	0.33
After 6 months	24	4.64	0.59	-5.738	0.000	-0.53	0.45

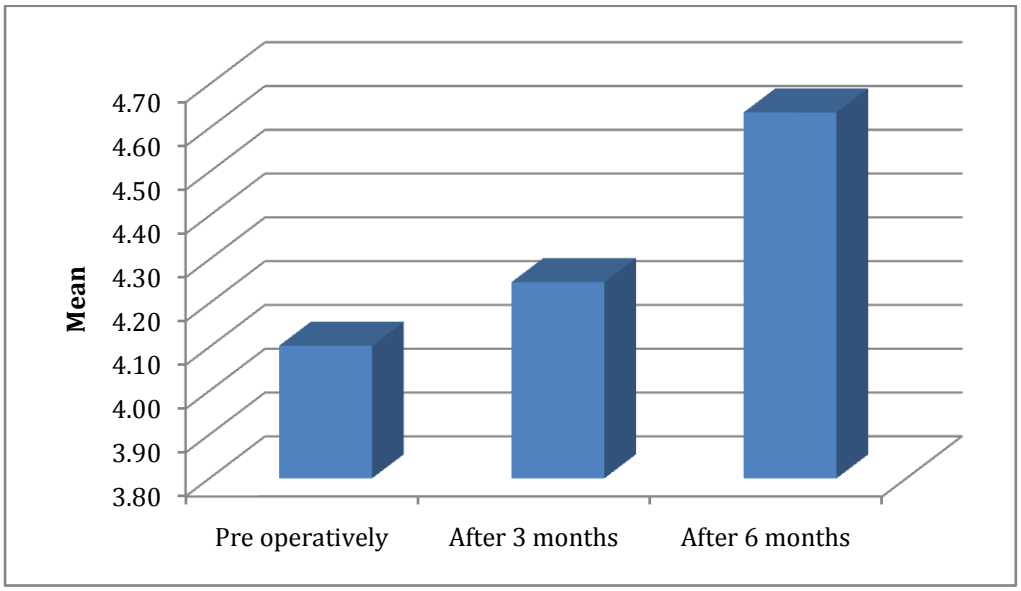


FIGURE 9: DIAGRAMMATIC REPRESENTATION OF MEAN ALBUMIN LEVELS

FIBROSCAN

Fibroscan was done of all the 24 patients undergoing bariatric surgery at the time of admission and at interval of 6 months post-operatively.

Table 12 and Figure 10 shows that among 24 patients fibroscan done preoperatively depicted the fibrosis gradeas F0 in 7 patients (29%), F1 in 2 patients (8.3%), F2 in 4 patients (17%),F3 in 7 patients (29%) , F4 in 4 patients (16.7%) .

TABLE 12: FIBROSIS GRADE USING FIBROSCAN AT ADMISSION

Fibrosis grade	Number of patients (%) (n=24)
F0	7 (29%)
F1	2 (8.3%)
F2	4 (16.7%)
F3	7 (29%)
F4	4 (16.7%)

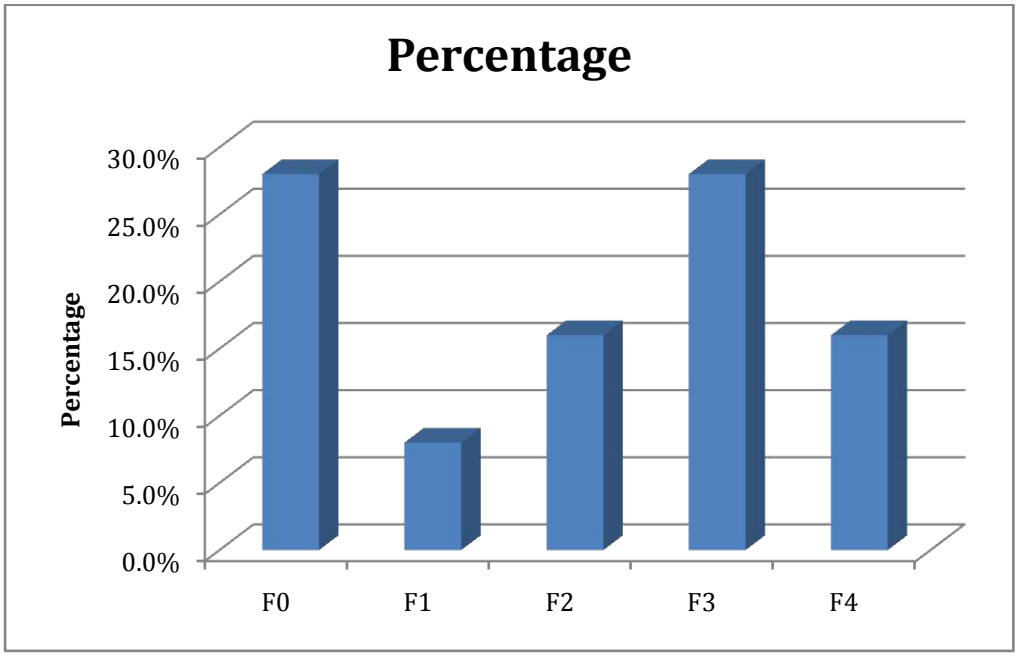


FIGURE 10: FIBROSIS GRADING USING FIBROSCAN PRE-OPERATIVELY

Table 13 and Figure 11 shows that among 24 patients fibroscan done post-operatively depicted the fibrosis gradeas F0 in 11 patients (46%), F1 in 6 patients (25%), F2 in 6 patients (25%), F3 in 1 patients (4%) which showed a significant fall in fibrosis grade among the patients.

TABLE 13: FIBROSIS GRADE USING FIBROSCAN AT 6 MONTHS INTERVAL

Fibrosis grade	Number of patients (%)
F0	11 (46%)
F1	6 (25%)
F2	6 (25%)
F3	1 (4%)
F4	0

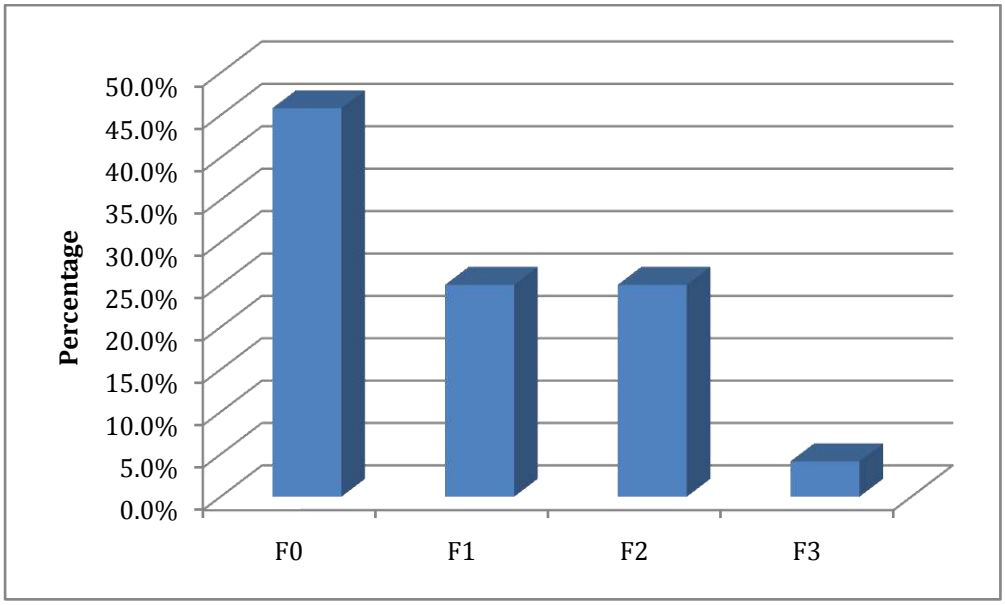


FIGURE 11: FIBROSIS GRADING USING FIBROSCAN AT 6 MONTHS POST-OPERATIVELY

In our study the CAP assesment using fibroscan was done pre and post-operatively at 6 months of time interval. Minimum CAP value recorded pre-operatively using Fibroscan was 281db/m and maximum was 402db/m. Table 14.shows median CAP value of 371.79 of the 24 patients pre-operatively and 297.29 post-operatively at 6 months interval. Which showed a significant fall in CAP value ($p<0.05$) after the bariatric surgery.

TABLE 14: MEAN CAP VALUE OF PATIENT PRE AND POST OPERATIVELY AFTER BARIATRIC SURGERY

FIBROSCAN				Z	p-value	Difference	
	N	Mean	SD			Mean	SD
FIBROSCAN Pre operatively	24	371.79	35.74				
FIBROSCAN post operative	24	297.29	28.48	20.867	0.000	74.50	17.49

NAFLD Fibrosis Score

NAFLD Fibrosis Score were calculated at the time of admission , at interval of 3 months and 6 months of the 24 patients who underwent bariatric surgery as shown in Table 15.

TABLE 15: NAFLD FIBROSIS SCORE SEVERITY AND NUMBER OF PATIENTS

Fibrosis severity	**On admission (no.of patients)**	**At 3 months (no.of patients)**	**At 6 months (no.of patients)**
F0-F2	5	12	18
INDETERMINATE SCORE	15	11	6
F3-F4	4	1	0

Table 16 and figure 12 shows, There was significant reduction (p-value <0.05) in NAFLD Fibrosis score post-operatively, with the mean NAFLD Fibrosis score decreasing from -0.54 preoperatively (at the time of admission) to -1.50 at 3 months and -2.36 at 6 months post operatively.

TABLE 16: NAFLD FIBROSIS SCORE PRE AND POST-OPERATIVELY AFTER BARIATRIC SURGERY

NAFLD FIBROSIS				t	p-value	Difference	
	N	Mean	SD			Mean	SD
Pre operatively	24	-0.54	1.61				
After 3 months	24	-1.50	1.20	5.315	0.000	0.96	0.88
After 6 months	24	-2.36	1.25	11.522	0.000	1.82	0.77

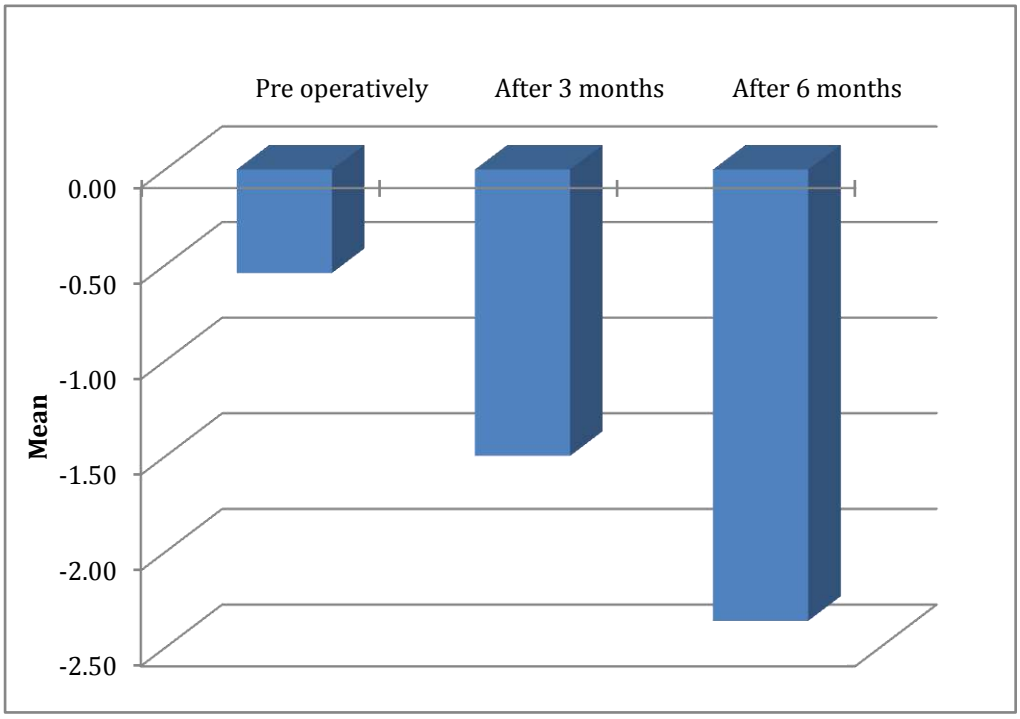


FIGURE 12: DIAGRAMMATIC REPRESENTATION OF MEAN NAFLD FIBROSIS SCORE

DISCUSSION

Bariatric – associated weight loss mechanism and NAFLD

NAFLD has become an epidemic of sizable proportions. Not surprisingly the rapid increase in the prevalence of NAFLD has paralleled the global rise in obesity, type II DM, and metabolic syndrome, known major risk factors for NAFLD.[4,55] Currently bariatric surgery provides the highest level of excess weight loss compared with other available methods achieving approximately 62% with gastric bypass, 4% with laparoscopic adjustable gastric banding, and 66% with laparoscopic sleeve gastrectomy.[56,57] There are several potential mechanistic pathways through which bariatric surgery is likely to have potential benefits in ameliorating factors, such as insulin resistance, lipid profile, inflammation, weight loss, and adipokinesthat contribute markedly to pathogenesis of NAFLD.[58] The mechanism of how bariatric surgery plays a role as potential treatment of NAFLD is complex and not fully understood. Firstly, bariatric surgery is associated with sustained and significant weight loss.[14] Second, it leads to improvement of insulin sensitivity, normoglycemia, improved dyslipidemia, and resolution of hypertension , all of which play a role in pathophysiology of steatosis and probably trigger steatohepatitis and fibrosis.[59,60] Thirdly, it reduces obesity related low grade chronic inflammation resulting from excess production of cytokines like tumor necrosis factor-α, interleukin-1, interleukin-8, interleukin-18, monocyte chemoattractant protein-1, and C-reactive protein.[61-63] Last it acts by producing complex hormonal changes in ghrelin, glucagon like peptide-1, peptide YY , oxyntomodulin , adiponectin, etc.[64,65]

Liver biopsy is the current gold standard for the diagnosis and assessment of severity of liver fibrosis; however, it is an invasive procedure with limitations such as sampling variability, expense and associated complications.[31] These limitations have led to the development of newer, simpler and less invasive methods of assessing liver fibrosis. The NAFLD fibrosis score was first described by Angulo et al. in 2007 [66] and is a simple non-invasive tool used for liver fibrosis

assessment. It comprises a composite score of age, hyperglycaemia, body mass index, platelet count, albumin, aspartateaminotransferase (AST) and alanineaminotransferase (ALT). In the 2007 paper by Angulo et al., routine demographic, clinical and laboratory values were analysed using multivariate modelling to predict the presence or absence of advanced fibrosis and the above six variables were found to be independent indicators of advanced liver fibrosis. The NAFLD fibrosis score is the most studied score with external validation in multiple studies and results confirmed with a meta-analysis by Musso et al.[67] Other studies have also shown that the NAFLD fibrosis score has an acceptable sensitivity, specificity and positive and negative predictive values for advanced liver fibrosis of 100%, 83%, 63% and 100%, respectively.[68] A higher NAFLD fibrosis score is also shown to be a significant predictor of mortality in NAFLD patients, with an odds ratio of 2.6.[69] In our study 5/24 patients were lying in F0-F2 group , 15/24 were lying in indeterminate score group, and 4/24 were lying in F3-F4 group which was reduced to 18/24 in F0-F2 group , 6/24 in indeterminate group post-operatively at 6 months interval. Similar results were seen in Musso et al.[67] The use of a validated non-invasive scoring system allowed us to assess our patient population at multiple time intervals without subjecting them to potential risks that may arise from multiple liver biopsies.

Fibroscan is an emerging tool for non-invasive assessment of NAFLD. The advantage of Transient Elastography includes assessment of an area of hundred times bigger than a biopsy sample, and good correlation with hepatic steatosis and fibrosis. Previous studies have reported that Fibroscan correlates with fibrosis and hepatic steatosis in patient with Hepatitis B virus, Hepatitis C virus. There is limited data about the usefulness of fibroscan in morbidly obese patients.[70-73] In our study success rate was similar to that reported by Naveauet al.[70] However, the fibroscan success rate reported by Weiss et al [71] was lower (63%) compared with our study . There was a significant decline in CAP values 6 months after surgery, the fibrosis grade after remained low (F0/F1) in most of the patients. Similarly, in our study 25% (6/24)

had grade 2 and 4% (1/25) grade 3 fibrosis, while all other patient had grade 0 46%(11/24) or grade 1 25%(6/24) fibrosis in postoperative period. The advance fibrosis grade that was F4was present in 4 patients preoperatively, among these 4 patients F4 grade was reduced to F2 in 3 patients and to F3 in 1 patient postoperatively at 6 months. The difference in fibrosis in postoperative period could be because of heterogeneity in study population and study designs, variety of bariatric procedures performed.

In this study, we analyzed the association of clinical factors, which included age; gender; BMI; type2 diabetes mellitus; hypertension; metabolic syndrome as a whole; and laboratory values of ALT, AST, ALP, HDL, and TGs of morbidly obese patients after 3 months and at 6 months post operatively.

We found that 71% of patients were female and 29% were males. And it was seen, that 41% of patients were in age group more than 60 years. We could not find any statistically significant association of age and gender with the occurrence of NAFLD. While some studies show a higher risk in men, others report a higher risk in women and some report no association as in our study.[3] Older age is reported in some studies to be associated with a higher risk of NASH, fibrosis, and cirrhosis , We did not find significant association of NAFLD with increasing age in our study. Many studies have shown a lack of an association with increasing BMI and the prevalence of NAFLD.[59,70]

Our study had 42% of diabetic patients. There was a significant association between TGs, LDL, HDL and HbA1c after 6 months post operatively. Insulin resistance has been shown to play a key role in this pathophysiology probably triggering steatohepatitis and fibrosis in these patients. Frank DM most probably acts by causing variability of blood glucose which might induce monocyte adhesion to endothelial cells, activate inflammatory cytokines and inflammation, and increase oxidative stress in the liver of patients with NAFLD leading to the histological severe forms.[9,17]

It has been shown that liver enzyme levels in NAFLD fluctuate with normal values being present in up to 78% of patients at any one

time.[10] When levels are elevated, the increase is mild and often restricted to one or both of alanine aminotransferase (ALT) and aspartate aminotransferase (AST). In our study, there was mean deviation of 5.45 in SGOT levels, after 6 months post op and SGPT showed mean deviation of 8.14 post-op after 6 months. As majority of the patients had normal liver enzymes, we analyzed the association of NAFLD and its highrisk forms with the mean levels of liver enzymes without using cutoffs. Higher mean levels of ALT were found to be the only laboratory parameter that correlated independently with a diagnosis of NAFLD and NASH. NAFLD was associated with elevated levels of AST though this was not statistically significant. NASH was also significantly associated with elevated levels of AST though these were not found to be an independent predictor of NASH. This finding supports the findings of several studies that have demonstrated that increased concentrations of hepatic enzyme in serum, even within the reference interval, may be related to increased risk of NAFLD and NASH.

In this study, bariatric surgery was not intended to treat NAFLD. Patients with more severe liver disease might have not been referred for bariatric consultation, and it is not known if these patients were more likely to develop progressive liver disease after weight loss, thus resulting in a selection bias. However, the findings of our study provide indirect evidence for the use of bariatric surgery in patients with morbid obesity and histologic severe forms of NAFLD.

Limitation of our study was a short-term follow up, we could not do follow up for a longer period of time, long term follow up for approximately 1 year might have shown further improvement in NAFLD staging.

SUMMARY AND CONCLUSION

The present prospective study was conducted in the department of General surgery of The Dayanand Medical College & Hospital, Ludhiana. The study was done in morbidly obese patients to compare the preoperative and postoperative effect of Bariatric surgery on NAFLD status of morbidly obese patients**.** A total of 24 patients who were fulfilling the criteria of bariatric surgery was taken for the study. A written informed consent was taken from every patient. Institutional ethical committee approved the study.

Various findings were observed in our study:

1. The fibrosis score observed using Fibroscan showed significant improvement. Patients lying in F0-F1 group were 9/24 preoperatively which was improved to 17/24 postoperatively at 6 months interval. Patients lying in F3-F4 group were 11/24 preoperatively which was reduced to 1/24 postoperatively at 6 months interval.
2. CAP assessment done using Fibroscan showed significant improvement preoperatively and postoperatively at 6 months interval. Median CAP value of 371.79 of the 24 patients was reduced to 297.29 postoperatively.
3. NAFLD Fibrosis score showed significant improvement. Patients lying in F0-F2 group were 5/24 at the time of admission that was improved to 12/24 and 18/24 at 3 months and 6 months interval respectively. While patient lying in F3-F4 group 4/24 at the time of admission was reduced to 1/24 at 3 months interval.

Thus our study showed that bariatric surgery is related to significant improvement of NAFLD status in morbidly obese patients.

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Sr. No	Date	Name	OPD/ WARD	Age	Sex	CR No	DOA	DOD	History of present illness	Past history	Medical treatment for obesity	Any other therapy for obesity		Other comorbidities						
												Diet therapy duration (Kcal/day)	Physical therapy	Hypertension	CAD	History of DM	Blood Pressure (mm/Hg)	Temperature (F)	SpO2 (%) (on room air)	Pulse Rate (beats/min)
1	Jan-19	JK	WARD	45 YRS	FEMALE	617701	29-Dec-19	1-Jan-19	MORBID OBESITY	HYPOTHYROIDISM & DEPRESSION	NO	NO	NO	NO	NO	NO	140/80	98.4	97	80
2	Jan-19	RK	WARD	32 YRS	FEMALE	635050	1/1/2019	4/1/2019	MORBID OBESITY	NO	NO	YES	NO	NO	NO	NO	120/70	98.4	96	78
3	Jan-19	KK	WARD	36 YRS	FEMALE	648305	4/1/2019	8/1/2019	MORBID OBESITY	HYPERTENSION	NO	NO	NO	YES	NO	NO	140/80	98.4	96	108
4	Jan-19	JK	WARD	33 YRS	FEMALE	624071	21/1/2019	24/1/2019	MORBID OBESITY	HYPOTHYROIDISM & BRONCHIAL ASTHMA	NO	NO	NO	NO	NO	NO	130/76	98.4	95	86
5	Mar-19	RB	WARD	62 YRS	FEMALE	681995	6/3/2019	9/3/2019	MORBID OBESITY WITH BREATHLESSNESS	HYPERTENSION & DEPRESSION/ TKR 2009 RIGHT SIDE	NO	NO	NO	YES	NO	YES	150/80	98.4	96	82
6	Mar-19	JK	WARD	60 YRS	FEMALE	913142	9/3/2020	12/3/2020	MORBID OBESITY	TYPE-II DM	NO	NO	NO	NO	NO	YES	112/60	98.6	96	94
7	Apr-19	AR	WARD	52 YRS	FEMALE	704663	8/4/2019	11/4/2020	MORBID OBESITY WITH BREATHLESSNESS	NO	NO	NO	NO	NO	NO	NO	130/80	98.4	96	96
8	May-19	VR	WARD	26 YRS	FEMALE	732278	20/5/2019	23/5/2020	MORBID OBESITY WITH BREATHLESSNESS	NO	NO	YES	NO	NO	NO	NO	130/76	98.4	97	82
9	Jun-19	OP	WARD	74 YRS	MALE	36331	10/6/2019	13/6/2019	MORBID OBESITY WITH BREATHLESSNESS	NO	NO	NO	NO	NO	YES	NO	130/74	97.6	98	84
10	Jun-19	SS	WARD	38 YRS	MALE	743901	14/6/2019	17/6/2019	MORBID OBESITY	NO	NO	YES	NO	NO	NO	NO	130/80	98.4	97	82
11	Jun-19	NK	WARD	45 YRS	FEMALE	752727	21/6/2019	25/6/2019	MORBID OBESITY WITH BREATHLESSNESS	NO	NO	NO	NO	NO	NO	NO	130/80	98.4	95	96
12	Jul-19	HK	WARD	54 YRS	FEMALE	760282	3/7/2019	7/7/2019	MORBID OBESITY WITH BREATHLESSNESS	NO	NO	NO	NO	NO	NO	YES	130/80	98.4	96	82
13	Jul-19	JS	WARD	35 YRS	MALE	719448	8/7/2019	12/7/2019	MORBID OBESITY WITH BREATHLESSNESS	NO	NO	YES	NO	NO	NO	NO	130/80	98.4	96	84
14	Jul-19	SS	WARD	43 YRS	MALE	759333	11/7/2019	14/7/2019	MORBID OBESITY	NO	NO	NO	NO	NO	NO	NO	130/80	98.4	96	82

Sr. No	Date	Name	OPD/ WARD	Age	Sex	CR No	DOA	DOD	History of present illness	Past history	Medical treatment for obesity	Any other therapy for obesity		Other comorbidities						
												Diet therapy duration (Kcal/day)	Physical therapy	Hypertension	CAD	History of DM	Blood Pressure (mm/Hg)	Temperature (F)	SpO2 (%) (on room air)	Pulse Rate (beats/min)
15	Sep-19	HS	WARD	44 YRS	FEMALE	182910	2/9/2019	4/9/2019	MORBID OBESITY WITH BREATHLESSNESS	NO	NO	NO	NO	NO	NO	NO	130/80	97.6	96	96
16	Sep-19	JP	WARD	58 YRS	MALE	812053	17/9/2019	20/9/2019	MORBID OBESITY	NO	NO	NO	NO	NO	NO	YES	130/80	98.4	97	82
17	Sep-19	AG	WARD	31 YRS	MALE	505192	18/9/2019	21/9/2019	MORBID OBESITY	TYPE-II DM	NO	NO	NO	NO	NO	YES	130/80	98.4	98	84
18	Nov-19	NQ	WARD	40 YRS	FEMALE	851443	18/11/2019	22/11/2019	MORBID OBESITY	NO	NO	NO	NO	YES	NO	NO	130/80	98.4	96	88
19	Nov-19	RS	WARD	51 YRS	FEMALE	852433	19/11/2019	22/11/2019	MORBID OBESITY	NO	NO	NO	NO	YES	NO	YES	130/80	98.4	97	96
20	Dec-19	GG	WARD	39 YRS	MALE	867708	16/12/2019	19/12/2019	MORBID OBESITY	NO	NO	NO	NO	NO	NO	YES	130/80	98.4	97	82
21	Feb-20	AA	WARD	34 YRS	FEMALE	651112	19/2/2020	21/2/2020	MORBID OBESITY	NO	NO	NO	NO	NO	NO	NO	110/60	97.6	95	104
22	Feb-20	IK	WARD	51 YRS	FEMALE	900644	17/2/2020	24/2/2020	MORBID OBESITY	TYPE-II DM/	NO	NO	NO	NO	NO	YES	120/90	98.2	96	82
23	Feb-20	KK	WARD	57 YRS	FEMALE	679675	13/2/2020	17/2/2020	MORBID OBESITY	HYPERTENSION	NO	NO	NO	YES	NO	NO	130/80	98.2	96	88
24	May-20	AK	WARD	53 YRS	FEMALE	927968	28/5/2020	1/6/2020	MORBID OBESITY	TYPE-II DM	NO	NO	NO	NO	NO	YES	122/64	98.4	95	92

Sr. No	Date	Name	General physical examination									Significant systemic examination					Blood Pressure (Systolic/ Diastolic) (mm Hg)		
			Respiratory Rate (breaths/min)	Pallor	Cyanosis	Lymphadenopathy	Clubbing	Edema	Icterus	Petechiae/ purpura	Others	Chest	Per abdomen	CVS	CNS	Investigations	Pre operatively	After 3 months	After 6 months
1	Jan-19	JK	20	NO	NO	NO	NO	NO	NO	NO	NO	B/L A/E PRESENT; NORMAL VESICULAR BREATH SOUNDS	SOFT, NON TENDER, BOWEL SOUNDS+	NORMAL S1 S2 HEARD	E4V5M6; B/L PUPIL REACTING TO LIGHT	2D-ECHO-NORMAL LV SIZE & SYSTOLIC FUNCTION; LVEF-60%	140/90	140/90	140/80
2	Jan-19	RK	20	NO	NO	NO	NO	NO	NO	NO	NO	B/L A/E PRESENT; NORMAL VESICULAR BREATH SOUNDS	SOFT, NON TENDER, BOWEL SOUNDS+	NORMAL S1 S2 HEARD	E4V5M6; B/L PUPIL REACTING TO LIGHT	NO	120/70	130/70	130/70
3	Jan-19	KK	16	NO	NO	NO	NO	NO	NO	NO	NO	B/L A/E PRESENT; NORMAL VESICULAR BREATH SOUNDS	SOFT, NON TENDER, BOWEL SOUNDS+	NORMAL S1 S2 HEARD	E4V5M6; B/L PUPIL REACTING TO LIGHT	NO	140/80	140/90	140/90
4	Jan-19	JK	20	NO	NO	NO	NO	NO	NO	NO	NO	B/L A/E PRESENT; NORMAL VESICULAR BREATH SOUNDS	SOFT, NON TENDER, BOWEL SOUNDS+	NORMAL S1 S2 HEARD	E4V5M6; B/L PUPIL REACTING TO LIGHT	PFT-FVC-76%; FEV1-59%; FEV1/FVC-82%;VC-76%	130/70	130/70	130/70
5	Mar-19	RB	18	NO	NO	NO	NO	NO	NO	NO	NO	B/L A/E PRESENT; NORMAL VESICULAR BREATH SOUNDS	SOFT, NON TENDER, BOWEL SOUNDS+	NORMAL S1 S2 HEARD	E4V5M6; B/L PUPIL REACTING TO LIGHT	2D-ECHO-NORMAL LV SIZE & SYSTOLIC FUNCTION; LVEF-60% ; PFT- FEV1-89%;VC-91%	150/80	130/70	130/70
6	Mar-19	JK	22	NO	NO	NO	NO	NO	NO	NO	NO	B/L A/E PRESENT; NORMAL VESICULAR BREATH SOUNDS	SOFT, NON TENDER, BOWEL SOUNDS+	NORMAL S1 S2 HEARD	E4V5M6; B/L PUPIL REACTING TO LIGHT	2D-ECHO-NORMAL LV SIZE & SYSTOLIC FUNCTION; LVEF-60%	130/80	134/80	120/80
7	Apr-19	AR	18	NO	NO	NO	NO	NO	NO	NO	NO	B/L A/E PRESENT; NORMAL VESICULAR BREATH SOUNDS	SOFT, NON TENDER, BOWEL SOUNDS+	NORMAL S1 S2 HEARD	E4V5M6; B/L PUPIL REACTING TO LIGHT	NO	130/80	130/70	130/70
8	May-19	VR	18	NO	NO	NO	NO	NO	NO	NO	NO	B/L A/E PRESENT; NORMAL VESICULAR BREATH SOUNDS	SOFT, NON TENDER, BOWEL SOUNDS+	NORMAL S1 S2 HEARD	E4V5M6; B/L PUPIL REACTING TO LIGHT	NO	130/76	130/70	130/70
9	Jun-19	OP	20	NO	NO	NO	NO	NO	NO	NO	NO	B/L A/E PRESENT; NORMAL VESICULAR BREATH SOUNDS	SOFT, NON TENDER, BOWEL SOUNDS+	NORMAL S1 S2 HEARD	E4V5M6; B/L PUPIL REACTING TO LIGHT	2D-ECHO-CAD/RWMA/SEVERE LV SYSTOLIC DYSFUNCTION; EF-28%; MILD MR; MILD TR WITH PASP-40mmHg; LV DIASTOLIC DYFUNCTION (TYPE-III)	120/70	130/70	130/70
10	Jun-19	SS	20	NO	NO	NO	NO	NO	NO	NO	NO	B/L A/E PRESENT; NORMAL VESICULAR BREATH SOUNDS	SOFT, NON TENDER, BOWEL SOUNDS+	NORMAL S1 S2 HEARD	E4V5M6; B/L PUPIL REACTING TO LIGHT	2D-ECHO-NORMAL LV SIZE & SYSTOLIC FUNCTION; LVEF-60% ; PFT-FVC-71%; FEV1-68%; FEV1/FVC-99%;VC-65% MILD RESTRICTION	120/70	130/70	130/70
11	Jun-19	NK	20	NO	NO	NO	NO	NO	NO	NO	NO	B/L A/E PRESENT; NORMAL VESICULAR BREATH SOUNDS	SOFT, NON TENDER, BOWEL SOUNDS+	NORMAL S1 S2 HEARD	E4V5M6; B/L PUPIL REACTING TO LIGHT	NO	130/70	130/70	130/70
12	Jul-19	HK	20	NO	NO	NO	NO	NO	NO	NO	NO	B/L A/E PRESENT; NORMAL VESICULAR BREATH SOUNDS	SOFT, NON TENDER, BOWEL SOUNDS+	NORMAL S1 S2 HEARD	E4V5M6; B/L PUPIL REACTING TO LIGHT	2D-ECHO-NORMAL LV SIZE & SYSTOLIC FUNCTION; LVEF-60%	130/70	130/70	130/70
13	Jul-19	JS	20	NO	NO	NO	NO	NO	NO	NO	NO	B/L A/E PRESENT; NORMAL VESICULAR BREATH SOUNDS	SOFT, NON TENDER, BOWEL SOUNDS+	NORMAL S1 S2 HEARD	E4V5M6; B/L PUPIL REACTING TO LIGHT	NO	130/70	130/70	130/70
14	Jul-19	SS	20	NO	NO	NO	NO	NO	NO	NO	NO	B/L A/E PRESENT; NORMAL VESICULAR BREATH SOUNDS	SOFT, NON TENDER, BOWEL SOUNDS+	NORMAL S1 S2 HEARD	E4V5M6; B/L PUPIL REACTING TO LIGHT	2D-ECHO-POST DVR-PROSTHETIC (METALLIC) MITRAL & AAORTIC VALVE NORMAL FUNCTION; NORMAL LV SYSTOLIC FUNCTION;EF-60%;LA DILATED; NO CLOT ; PFT-FVC-61%; FEV1-49%; FEV1/FVC-83%; SEVERE OBSTRUCTION	120/70	130/70	130/70

Sr. No	Date	Name	General physical examination									Significant systemic examination					Blood Pressure (Systolic/ Diastolic) (mm Hg)		
			Respiratory Rate (breaths/min)	Pallor	Cyanosis	Lymphadeno pathy	Clubbing	Edema	Icterus	Petechiae/ purpura	Others	Chest	Per abdomen	CVS	CNS	Investigations	Pre operatively	After 3 months	After 6 months
15	Sep-19	HS	20	NO	NO	NO	NO	NO	NO	NO	NO	B/L A/E PRESENT; NORMAL VESICULAR BREATH SOUNDS	SOFT, NON TENDER, BOWEL SOUNDS+	NORMAL S1 S2 HEARD	E4V5M6; B/L PUPIL REACTING TO LIGHT	2D-ECHO-NORMAL LV SIZE & SYSTOLIC FUNCTION; LVEF-60%	120/70	130/70	130/70
16	Sep-19	JP	20	NO	NO	NO	NO	NO	NO	NO	NO	B/L A/E PRESENT; NORMAL VESICULAR BREATH SOUNDS	SOFT, NON TENDER, BOWEL SOUNDS+	NORMAL S1 S2 HEARD	E4V5M6; B/L PUPIL REACTING TO LIGHT	NO	120/70	130/70	130/70
17	Sep-19	AG	20	NO	NO	NO	NO	NO	NO	NO	NO	B/L A/E PRESENT; NORMAL VESICULAR BREATH SOUNDS	SOFT, NON TENDER, BOWEL SOUNDS+	NORMAL S1 S2 HEARD	E4V5M6; B/L PUPIL REACTING TO LIGHT	NO	120/70	130/70	130/70
18	Nov-19	NQ	20	NO	NO	NO	NO	NO	NO	NO	YES	B/L A/E PRESENT; NORMAL VESICULAR BREATH SOUNDS	SOFT, NON TENDER, BOWEL SOUNDS+	NORMAL S1 S2 HEARD	E4V5M6; B/L PUPIL REACTING TO LIGHT	2D-ECHO-NORMAL LV SIZE & SYSTOLIC FUNCTION; LVEF-60%	130/70	130/70	130/70
19	Nov-19	RS	20	NO	NO	NO	NO	NO	NO	NO	NO	B/L A/E PRESENT; NORMAL	SOFT, NON TENDER,	NORMAL S1	E4V5M6; B/L PUPIL	2D-ECHO-NORMAL LV SIZE &	120/70	130/70	120/70
20	Dec-19	GG	20	NO	NO	NO	NO	NO	NO	NO	NO	B/L A/E PRESENT; NORMAL	SOFT, NON TENDER,	NORMAL S1	E4V5M6; B/L PUPIL	NO	120/70	130/70	120/70
21	Feb-20	AA	20	NO	NO	NO	NO	NO	NO	NO	NO	B/L A/E PRESENT; NORMAL	SOFT, NON TENDER,	NORMAL S1	E4V5M6; B/L PUPIL	2D-ECHO-NORMAL LV SIZE &	110/60	120/70	120/70
22	Feb-20	IK	24	NO	NO	NO	NO	NO	NO	NO	NO	B/L A/E PRESENT; NORMAL	SOFT, NON TENDER,	NORMAL S1	E4V5M6; B/L PUPIL	2D-ECHO-NORMAL LV SIZE &	120/70	130/90	120/80
23	Feb-20	KK	20	NO	NO	NO	NO	NO	NO	NO	NO	B/L A/E PRESENT; NORMAL	SOFT, NON TENDER,	NORMAL S1	E4V5M6; B/L PUPIL	2D-ECHO-NORMAL LV SIZE &	140/90	140/90	130/70
24	May-20	AK	22	NO	NO	NO	NO	NO	NO	NO	NO	B/L A/E PRESENT; NORMAL	SOFT, NON TENDER,	NORMAL S1	E4V5M6; B/L PUPIL	2D-ECHO-NORMAL LV SIZE &	122/84	130/80	130/80

Sr. No	Date	Name	Height (m2)			Weight (Kg)			BMI (Kg/m2)			Hip/Waist ratio			Triglycerides			LDL			HDL		
			Preoperatively	After 3 months	After 6 months	Preoperatively	After 3 months	After 6 months	Preoperatively	After 3 months	After 6 months	Preoperatively	After 3 months	After 6 months	Preoperatively	After 3 months	After 6 months	Preoperatively	After 3 months	After 6 months	Preoperatively	After 3 months	After 6 months
1	Jan-19	JK	1.5	1.5	1.5	102	90	78	45.3	40	34.7	0.93	0.89	0.83	165	154	150	102	100	50	67	68	60
2	Jan-19	RK	1.6	1.6	1.6	100	89.6	81	37.2	35	31.6	0.86	0.82	0.8	38	35	38	113	100	72	54	53	36
3	Jan-19	KK	1.5	1.5	1.5	113	94	88	50.2	41.8	39.1	0.9	0.85	0.82	66	60	65	102	80	78	68	67	56
4	Jan-19	JK	1.7	1.7	1.7	170	140	122	58.8	48.4	42.2	0.93	0.91	0.87	125	122	122	102	99	48	57	56	50
5	Mar-19	RB	1.57	1.57	1.57	96	85	78	38.9	34.5	31.6	0.9	0.87	0.84	175	100	90	98	86	55	69	62	52
6	Mar-19	JK	1.6	1.6	1.6	114	98	94	44.5	38.3	36.7	0.88	0.86	0.83	74	55	60	78	66	60	38	39	38
7	Apr-19	AR	1.58	1.58	1.58	98	84	76	39.3	33.6	30.4	0.87	0.84	0.81	68	65	60	100	89	60	68	66	30
8	May-19	VR	1.6	1.6	1.6	92	84	80	35.9	32.8	31.3	0.89	0.85	0.83	90	88	80	73	65	51	42	42	38
9	Jun-19	OP	1.57	1.57	1.57	108	92	82	45.4	37.3	33.3	1.06	0.97	0.95	119	110	100	43	39	40	67	60	42
10	Jun-19	SS	1.46	1.46	1.46	172	130	100	78.7	61	46.9	1.09	1.03	0.97	347	244	147	171	168	138	58	50	55
11	Jun-19	NK	1.5		1.5	94	81	78	41.8	35.6	34.7	0.91	0.87	0.84	165	141	100	144	144	52	36	38	38
12	Jul-19	HK	1.57	1.57	1.57	100	80	75	40.6	35.6	33.3	0.98	0.93	0.85	650	355	302	173	122	61	47	46	39
13	Jul-19	JS	1.5	1.5	1.5	120	90	85	53.3	40	37.8	1.08	1	0.96	127	110	100	79	60	48	44	44	34
14	Jul-19	SS	1.56	1.56	1.56	96	82	78	39.4	35.6	34.7	1.09	1.02	0.97	167	150	88	107	99	53	39	30	31

Sr. No	Date	Name	Height (m2)			Weight (Kg)			BMI (Kg/m2)			Hip/Waist ratio			Triglycerides			LDL			HDL		
			Preoperatively	After 3 months	After 6 months	Preoperatively	After 3 months	After 6 months	Preoperatively	After 3 months	After 6 months	Preoperatively	After 3 months	After 6 months	Preoperatively	After 3 months	After 6 months	Preoperatively	After 3 months	After 6 months	Preoperatively	After 3 months	After 6 months
15	Sep-19	HS	1.5	1.5	1.5	107	93	89	47.6	41.3	39.6	0.96	0.93	0.88	102	92	78	78	65	56	40	33	35
16	Sep-19	JP	1.5	1.5	1.5	123	105	99	54.7	46.7	44	1.1	1	0.96	160	145	92	102	80	69	31	30	30
17	Sep-19	AG	1.6	1.6	1.6	135	98	89	52.7	38.3	34.8	1.08	1.07	0.98	203	186	100	98	80	74	31	30	31
18	Nov-19	NQ	1.6	1.6	1.6	102	91	85	39.8	35.5	31.6	0.97	0.94	0.86	145	100	89	107	82	55	43	44	30
19	Nov-19	RS	1.59	1.59	1.59	126.4	100	90	49.8	39.6	35.6	1.02	0.98	0.89	98	74	62	83	79	49	56	44	31
20	Dec-19	GG	1.5	1.5	1.5	120	100	95	53.3	39.6	37.6	1.09	1.06	0.98	219	113	91	73	69	56	38	40	35
21	Feb-20	AA	1.46	1.46	1.46	119	100	92	55.8	46.9	43.2	1	0.9	0.84	163	121	85	110	100	61	30	30	30
22	Feb-20	IK	1.5	1.5	1.5	120	101	81	53.3	44.9	36	0.94	0.89	0.85	160	140	82	100	85	70	30	30	30
23	Feb-20	KK	1.6	1.6	1.6	127	100	90	49.6	39.1	35.8	0.89	0.84	0.82	116	128	100	80	78	76	34	33	34
24	May-20	AK	1.62	1.62	1.62	115	100	89	43.8	38.1	33.9	0.91	0.87	0.84	89	72	70	75	75	75	40	40	35

Sr. No	Date	Name	HbA1c			Fasting Glucose			SGOT (U/L)			SGPT (U/L)			PLATELETS (X 109/L)			ALBUMIN (g/dL)			FIBROSCAN (CAP SCORE/FIBROSIS GF		
			Preoperatively	After 3 months	After 6 months	Preoperatively	After 3 months	After 6 months	Preoperatively	After 3 months	After 6 months	Preoperatively	After 3 months	After 6 months	Preoperatively	After 3 months	After 6 months	Preoperatively	After 3 months	After 6 months	Preoperatively (db/m)		Postoperatively
1	Jan-19	JK	5.8	5.2	5.5	100	100	92	22	22	24	22	21	20	321	310	300	4.7	4.2	4.6	301	F0	246
2	Jan-19	RK	5.9	5.4	5	98	100	100	22	24	28	28	24	26	363	304	447	4.7	4.3	4.47	281	F0	234
3	Jan-19	KK	5.8	5.8	5.5	80	100	92	19	21	24	28	30	33	285	360	335	4.1	4	4.4	288	F0	240
4	Jan-19	JK	5.3	5.3	5.2	125	92	120	20	22	22	16	19	24	349	279	350	4.7	4.1	4.2	401	F4	320
5	Mar-19	RB	8.1	7	6.1	120	144	100	39	25	23	41	30	34	317	333	352	4.2	4.5	5.1	370	F1	300
6	Mar-19	JK	8.6	7.7	6.5	190	148	117	18	27	24	18	24	18	344	340	300	3.39	3.94	3.8	400	F2	334
7	Apr-19	AR	6.2	6.2	6	122	120	125	31	35	30	33	28	20	233	350	265	3.94	4.3	4.9	348	F0	268
8	May-19	VR	5.8	5.2	5.1	131	133	134	29	26	22	42	35	40	306	280	322	4.15	4.06	4.19	388	F2	310
9	Jun-19	OP	6.3	6.4	5	164	150	156	27	25	35	21	26	24	180	300	225	4.46	4.32	5.2	374	F3	280
10	Jun-19	SS	6.3	6	5.4	134	100	150	27	30	25	40	32	29	264	340	276	3.89	4.14	4.9	398	F4	326
11	Jun-19	NK	5.9	5.8	5.1	155	132	116	30	22	26	40	36	38	266	188	268	4.06	4.6	5.5	402	F3	328
12	Jul-19	HK	8	7.9	6.6	192	149	110	30	24	28	20	28	20	202	244	288	5	4.7	5.8	398	F3	288
13	Jul-19	JS	5.2	5.3	5.1	131	122	100	19	26	20	18	22	19	262	208	300	4.5	5	5.2	378	F2	300
14	Jul-19	SS	5.6	5.4	5	143	126	114	37	31	28	30	28	20	255	302	285	4.16	4.32	4.8	399	F4	288

Sr. No	Date	Name	HbA1c			Fasting Glucose			SGOT (U/L)			SGPT (U/L)			PLATELETS (X 109/L)			ALBUMIN (g/dL)			FIBROSCAN (CAP SCORE/FIBROSIS GF		
			Preoperatively	After 3 months	After 6 months	Preoperatively	After 3 months	After 6 months	Preoperatively	After 3 months	After 6 months	Preoperatively	After 3 months	After 6 months	Preoperatively	After 3 months	After 6 months	Preoperatively	After 3 months	After 6 months	Preoperatively (db/m)		Postoperatively
15	Sep-19	HS	5.8	5.8	5.6	188	130	92	29	32	28	34	32	20	202	290	230	3.5	3.94	3.8	376	F3	327
16	Sep-19	JP	9.1	7.6	6.4	190	136	98	51	32	40	59	42	24	225	210	256	3.8	4.2	4.4	388	F3	306
17	Sep-19	AG	9.6	8.2	6.5	225	199	100	30	34	38	50	32	40	223	249	227	4.17	4.6	5.1	358	F2	288
18	Nov-19	NQ	5.9	5.4	6	147	150	125	47	40	34	43	36	38	203	228	218	3.85	3.99	4.3	400	F3	330
19	Nov-19	RS	6.9	5.9	5.9	200	164	140	18	28	32	17	20	20	348	316	350	4.04	4.3	4.9	400	F4	334
20	Dec-19	GG	6.5	6	6.2	282	170	100	47	36	34	63	41	36	198	208	191	4.46	4.78	5.6	398	F3	304
21	Feb-20	AA	6.2	6	6.1	210	190	124	14	18	20	16	22	20	457	380	450	3.91	4.3	4.4	349	F0	298
22	Feb-20	IK	9.2	7.8	6.5	198	180	115	31	26	30	40	32	35	225	276	301	3.8	3.94	4.02	368	F0	288
23	Feb-20	KK	6.2	6	5.3	100	100	120	37	33	30	40	31	40	266	243	217	3.39	3.66	3.8	372	F0	300
24	May-20	AK	8.2	7.1	6.2	200	99	150	30	24	30	42	30	34	260	238	262	3.6	3.76	3.88	388	F1	298

Sr. No	Date	Name	(ADE)	NAFLD FIBROSIS SCORE						ANTI-HCV	Hbs Ag	USG Abdomen	Surgery Performed
			(db/m)	Preoperatively		After 3 months		After 6 months		Preoperatively	Preoperatively	Preoperatively	
1	Jan-19	JK	F0	-2.04	F0-F2	-2.01	F0-F2	-2.5	F0-F2	Non-reactive	Non-reactive	FATTY LIVER GRADE-II	MINI GASTRIC BYPASS
2	Jan-19	RK	F0	-4.04	F0-F2	-3.31	F0-F2	-5.1	F0-F2	Non-reactive	Non-reactive	FATTY LIVER GRADE-I	SLEEVE GASTRECTOMY
3	Jan-19	KK	F0	-1.36	INDETERMINANT SCORE	-3.04	F0-F2	-3.21	F0-F2	Non-reactive	Non-reactive	FATTY LIVER GRADE-III	SLEEVE GASTRECTOMY
4	Jan-19	JK	F2	-1.33	INDETERMINANT SCORE	-1.09	INDETERMINANT SCORE	-2.9	F0-F2	Non-reactive	Non-reactive	HEPATOMEGALY WITH FATTY LIVER GRADE-I	MINI GASTRIC BYPASS
5	Mar-19	RB	F0	-1.68	F0-F2	-2.61	F0-F2	-3.68	F0-F2	Non-reactive	Non-reactive	FATTY LIVER GRADE-II	MINI GASTRIC BYPASS
6	Mar-19	JK	F0	0.14	INDETERMINANT SCORE	-0.61	INDETERMINANT SCORE	-1.09	INDETERMINANT SCORI	Non-reactive	Non-reactive	FATTY LIVER GRADE-I	MINI GASTRIC BYPASS
7	Apr-19	AR	F0	-0.76	INDETERMINANT SCORE	-3.02	F0-F2	-2.51	F0-F2	Non-reactive	Non-reactive	FATTY LIVER GRADE-I	MINI GASTRIC BYPASS
8	May-19	VR	F0	-3.34	F0-F2	-3.21	F0-F2	-4.18	F0-F2	Non-reactive	Non-reactive	FATTY LIVER GRADE-II	MINI GASTRIC BYPASS
9	Jun-19	OP	F1	1.32	F3-F4	-1.23	INDETERMINANT SCORE	-0.72	INDETERMINANT SCORI	Non-reactive	Non-reactive	FATTY LIVER GRADE-III	SLEEVE GASTRECTOMY
10	Jun-19	SS	F3	1.8	F3-F4	-0.76	INDETERMINANT SCORE	-1.83	F0-F2	Non-reactive	Non-reactive	HEPATOMEGALY WITH FATTY LIVER GRADE-III	MINI GASTRIC BYPASS
11	Jun-19	NK	F2	-1.44	INDETERMINANT SCORE	-1.55	F0-F2	-3.18	F0-F2	Non-reactive	Non-reactive	FATTY LIVER GRADE-III	MINI GASTRIC BYPASS
12	Jul-19	HK	F1	0.32	INDETERMINANT SCORE	-0.63	INDETERMINANT SCORE	-2.73	F0-F2	Non-reactive	Non-reactive	FATTY LIVER GRADE-II	MINI GASTRIC BYPASS
13	Jul-19	JS	F1	-0.71	INDETERMINANT SCORE	-1.45	INDETERMINANT SCORE	-3.12	F0-F2	Non-reactive	Non-reactive	FATTY LIVER GRADE-III	SLEEVE GASTRECTOMY
14	Jul-19	SS	F2	-1.23	INDETERMINANT SCORE	-2.41	F0-F2	-2.31	F0-F2	Non-reactive	Non-reactive	HEPATOMEGALY WITH FATTY LIVER GRADE-III	MINI GASTRIC BYPASS

Sr. No	Date	Name	(ADE)	NAFLD FIBROSIS SCORE						ANTI-HCV	Hbs Ag	USG Abdomen	Surgery Performed
			(db/m)	Preoperatively		After 3 months		After 6 months		Preoperatively	Preoperatively	Preoperatively	
15	Sep-19	HS	F2	0.32	INDETERMINANT SCORE	-1.55	F0-F2	-0.86	INDETERMINANT SCORI	Non-reactive	Non-reactive	FATTY LIVER GRADE-II	SLEEVE GASTRECTOMY
16	Sep-19	JP	F1	2.17	F3-F4	1.24	F3-F4	0.03	INDETERMINANT SCORI	Non-reactive	Non-reactive	FATTY LIVER GRADE-II	SLEEVE GASTRECTOMY
17	Sep-19	AG	F1	0.5	INDETERMINANT SCORE	-1.01	INDETERMINANT SCORE	-2.63	F0-F2	Non-reactive	Non-reactive	HEPATOMEGALY WITH FATTY LIVER GRADE-II	MINI GASTRIC BYPASS
18	Nov-19	NQ	F2	-0.55	INDETERMINANT SCORE	-1.36	INDETERMINANT SCORE	-2.01	F0-F2	Non-reactive	Non-reactive	FATTY LIVER GRADE-II	MINI GASTRIC BYPASS
19	Nov-19	RS	F2	-1.25	INDETERMINANT SCORE	-1.63	F0-F2	-2.64	F0-F2	Non-reactive	Non-reactive	FATTY LIVER GRADE-III	SLEEVE GASTRECTOMY
20	Dec-19	GG	F1	0	INDETERMINANT SCORE	-1.5	F0-F2	-1.94	F0-F2	Non-reactive	Non-reactive	HEPATOMEGALY WITH	SLEEVE GASTRECTOMY
21	Feb-20	AA	F0	-2.83	F0-F2	-3.16	F0-F2	-4.12	F0-F2	Non-reactive	Non-reactive	HEPATOMEGALY WITH	MINI GASTRIC BYPASS
22	Feb-20	IK	F0	1.88	F3-F4	0.17	INDETERMINANT SCORE	-0.99	INDETERMINANT SCORI	Non-reactive	Non-reactive	FATTY LIVER GRADE-I	SLEEVE GASTRECTOMY
23	Feb-20	KK	F0	0.64	INDETERMINANT SCORE	-0.41	INDETERMINANT SCORE	-0.79	INDETERMINANT SCORI	Non-reactive	Non-reactive	FATTY LIVER GRADE-II	MINI GASTRIC BYPASS
24	May-20	AK	F0	0.48	INDETERMINANT SCORE	0.21	INDETERMINANT SCORE	-1.62	F0-F2	Non-reactive	Non-reactive	FATTY LIVER GRADE-	MINI GASTRIC BYPASS

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Date 9/12/19

The Principal,
DMC, Ludhiana.

Subject: **Approval of Plan of Thesis.**

This is to inform you that the Faculty of Medical Sciences in its meeting held on 29.05.2019 has approved the plan of thesis of **Nikhil**, a student of your college for **MS General Surgery** along with the names of Supervisor/Co-Supervisor as under:-

Topic Plan of Thesis

A prospective study to see the impact of bariatric surgery on non alcoholic fatty liver disease (nafld) in morbidly obese patient

Supervisor
Dr. Ashish Ahuja
Assoc. Prof.,
Dept. of Surgery,
DMCH, Ludhiana.

Co-Supervisor
Dr. P. S. Nain
Prof.,
Dept. of Surgery,
DMCH, Ludhiana.

Dr. Varun Mehta
Assoc. Prof.,
Dept. of Gastroenterology,
DMCH, Ludhiana.

The student has to submit the four copies of thesis (along with PDF format) , to the Controller of Examinations, BFUHS, Faridkot, at least six months before the commencement of theory examination, if he/she is otherwise eligible to take the examination. He /She must directed to attach a photocopy of this letter with the forwarding letter while submitting thesis to the University along with the letter of approval of University for change of supervisor/Co-Supervisor, change of topic,(if any).

S P Singh

Dr. S P Singh MS(Oph)
Controller of Examinations

Note :

1. *If any discrepancy found regarding topic of thesis or Supervisor/Co-Supervisor, inform undersigned within one month after issue of this letter.*
2. *The student may submit the thesis as per the schedule already conveyed i.e for May/June examination 30th Nov of the preceding the year of examination and for Nov/Dec examinations 31st May of the year of examination.*

A PROSPECTIVE STUDY TO SEE THE IMPACT OF BARIATRIC SURGERY ON NON ALCOHOLIC FATTY LIVER DISEASE (NAFLD) IN MORBIDLY OBESE PATIENT

THESIS PROPOSAL

FOR APPROVAL OF SUBJECT OF THESIS TO BE SUBMITTED

IN PARTIAL FULFILMENT OF THE REQUIREMENTS

FOR THE DEGREE OF

M.S

IN

SURGERY

OF THE

BABA FARID UNIVERSITY OF HEALTH SCIENCES
FARIDKOT

AUGUST, 2018

DR. NIKHIL

DEPARTMENT OF SURGERY
DAYANAND MEDICAL COLLEGE AND HOSPITAL
LUDHIANA

ABSTRACT OF PLAN OF THESIS

Title	A PROSPECTIVE STUDY TO SEE THE IMPACT OF BARIATRIC SURGERY ON NON ALCOHOLIC FATTY LIVER DISEASE (NAFLD) IN MORBIDLY OBESE PATIENT.
For the degree of	M.S. in General Surgery
Name of the candidate	Dr. Nikhil
Supervisor	Dr. Ashish Ahuja, Associate Professor, Department of Surgery, DMC&H, Ludhiana
Co-supervisor	Dr P S Nain, Professor, Department of Surgery, DMC&H, Ludhiana Dr. Varun Mehta, Associate Professor, Department of Gastroenterology, DMCH, Ludhiana
Institution	Dayanand Medical College & Hospital, Ludhiana
University	Baba Farid University of Health Sciences, Faridkot

Introduction: Nonalcoholic fatty liver disease(NAFLD) affects over 80% of obese patients and is associated with the metabolic syndrome. NAFLD can progress into cirrhosis, which is associated to hepatocellular carcinoma and a liver transplant could be indicated. It represents the third cause of liver transplant, and it is expected to be first by 2025.Weight loss is strongly advocated as a central treatment for NAFLD, bariatric surgery is considered unique to obtain durable and permanent weight loss in morbid obesity and could reverse metabolic syndrome consequences as well as fatty liver disease.

Aim of Study: To evaluate the effects of bariatric surgery on non alcoholic fatty liver disease in morbidly obese patients

Materials & Methods: This prospective study will be conducted at Dayanand Medical College and Hospital, Ludhiana and include patients admitted for morbid obesity with nonalcoholic fatty liver disease from November 2018 to February 2020, using NAFLD fibrosis score and fibroscan.

Clinical Significance: In this study we aim to determine the pattern of improvement in NAFLD with weight loss in patients undergoing bariatric surgery

Keywords: Bariatric surgery, weight loss, fatty liver

(Signature)	(Signature)	(Signature)	(Signature)	(Signature)
Candidate	Co-supervisor	Co-supervisor	Supervisor	Principal

CERTIFICATE OF FACILITIES AVAILABLE

This is to certify that the facilities for work on the subject of thesis titled **"A PROSPECTIVE STUDY TO SEE THE IMPACT OF BARIATRIC SURGERY ON NON ALCOHOLIC FATTY LIVER DISEASE (NAFLD) IN MORBIDLY OBESE PATIENT"** exist in the Department of Surgery, DMC&H, Ludhiana and will be provided to the candidate. We will see that the data being included in the thesis is genuine and is collected by the candidate himself under our supervision and guidance.

Supervisor

Dr. Ashish Ahuja
Associate Professor
Department of Surgery
DMCH, Ludhiana

Co-Supervisor(s)

Dr. P S Nain
Professor
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Dr. Jaspal Singh
Professor & Head
Department of Surgery
DMCH, Ludhiana

Place : _____

Date : _____

APPROVAL PROFORMA
BY
RESEARCH & ETHICAL COMMITTEE
DAYANAND MEDICAL COLLEGE & HOSPITAL, LUDHIANA

Name of candidate	Dr. Nikhil
Department	Surgery
Topic of Thesis	A PROSPECTIVE STUDY TO SEE THE IMPACT OF BARIATRIC SURGERY ON NON ALCOHOLIC FATTY LIVER DISEASE (NAFLD) IN MORBIDLY OBESE PATIENT
Likely date of appearing in PG examination	MAY 2021
Date of enrollment	10th May, 2018
Name of Head of Department	Dr. Jaspal Singh
1) Supervisor **2) Co-Supervisor(s)**	Dr. Ashish Ahuja Dr. P S Nain Dr. Varun Mehta
Signature of Members of Research Committee with Stamp	**Signature of Members of Ethics Committee with Stamp**
1. HOD Pharmacology	1. HOD Physiology
2. HOD Surgery	2. HOD Pathology
3. HOD Pathology	3. HOD Pharmacology
4. HOD Medicine	4. HOD SPM
5. Dean Academics	5. HOD Medicine
6. Vice Principal	6. HOD Surgery
	7. HOD Obst. & Gynae
	8. Legal Expert
	9. Secretary, IEC
	10. Dean Academics
	11. Vice Principal
	12. Principal

Approved: Yes / No

Chairperson
Research Committee

Chairperson
Research Committee

Approved: Yes / ~~No~~

Chairperson
Ethics Committee

Chairperson
Ethics Committee

BABA FARID UNIVERSITY OF HEALTH SCIENCES, FARIDKOT

PROFORMA FOR REGISTRATION OF SUBJECT FOR DISSERTATION

1	NAME OF THE CANDIDATE AND ADDRESS	Dr. Nikhil H.no.377 , New civil lines , Back side green view park, Hoshiarpur
2	NAME OF THE INSTITUTE	DAYANAND MEDICAL COLLEGE AND HOSPITAL, LUDHIANA (PUNJAB)
3	COURSE OF STUDY AND SUBJECT	M.S. GENERAL SURGERY
4	DATE OF ADMISSION OF COURSE	10.05.2018
5	TITLE OF THE TOPIC	A PROSPECTIVE STUDY TO SEE THE IMPACT OF BARIATRIC SURGERY ON NON ALCOHOLIC FATTY LIVER DISEASE (NAFLD) IN MORBIDLY OBESE PATIENT

6. BRIEF RESUME OF THE INTENDED WORK

6.1 NEED FOR THE STUDY

Non-alcoholic fatty liver disease (NAFLD) is the most common cause of liver dysfunction worldwide, and obesity is a well-documented risk factor for the disease. It is highest in populations with preexisting metabolic conditions, such as obesity, T2DM, and dyslipidemia. NAFLD can progress into cirrhosis, which is associated to hepatocellular carcinoma and a liver transplant could be indicated. It represents the third cause of liver transplant, and it is expected to be first by 2025. Weight loss is strongly advocated as a central treatment for NAFLD. We aim to define the pattern of improvement in NAFLD.

While NAFLD has a multidimensional etiology, weight loss remains at the forefront of current therapies. Bariatric surgery has also been shown to reverse steatosis in up to 91% of patients and improve steatohepatitis in as many as 81.3% of patients.Additionally, total weight loss>10% has been shown to decrease hepatic steatosis, necroinflammation, and fibrosis, thereby potentially improving liver-related outcomes . Although dietary modification and exercise regimens can promote weight loss, a vast majority of patients are unable to achieve and maintain the necessary amount of weight loss related to meaningful clinical improvement. Bariatric surgery is the most effective and durable treatment for weight loss and obesity-related comorbidities, also there are some evidences that bariatric surgery lead to decreasing of liver fat content and Insulin Resistance and improvement of metabolic syndrome, even before significant weight loss. Other studies suggest that improvement of metabolic syndrome after bariatric surgery has correlation with amount of weight loss. Bariatric surgery potentially can

improve the metabolic syndrome, NAFLD/NASH and liver fibrosis.Current NAFLD and NASH treatments include weight loss obtained by life style modification and pharmaceuticals but have poor effects in patients with body mass index >35 kg/m

Bariatric surgery is the most reliable method for achieving substantial, sustained weight loss. This procedure is safe when performed by a skilled surgeon, and the benefits include reduced weight, improved quality of life, decreased obesity-related comorbidities, and increased life expectancy. The morbidity of NAFLD is significant as it may become the most common indication for liver transplantation within the next 5 years. In addition to known benefits of weight loss and diabetes resolution, bariatric surgery has the potential to halt and reverse disease progression and future controlled trials should be performed to further define its benefit in the treatment of NAFLD in morbidly obese patients

6.2 REVIEW OF LITERATURE

Nonalcoholic fatty liver disease (NAFLD) has seen a remarkable increase in clinical and epidemiologic interest in recent years due to an immense global burden of disease. NAFLD has an estimated prevalence of 19%, affecting 28.8 million people in the United States.[1] NAFLD is characterized by lipid deposition in hepatocytes with progressive hepatocellular degeneration, inflammation, and fibrotic parenchymal changes.[2] NAFLD may result in progressive liver injury and is associated with increased mortality from cardiovascular disease, malignancy, and liver failure.[3] Morbid obesity and metabolic syndrome are also strongly linked with NAFLD and co-occurring health issues likely related to obesity.

While NAFLD has a multidimensional etiology, weight loss remains at the forefront of current therapies. Recent data suggests that a>10% weight loss is associated with a significant decrease in hepatic steatosis, necroinflammation, and fibrosis.[4] Although dietary modification and exercise regimens can promote weight loss, a vast majority of patients are unable to achieve and maintain the necessary amount of weight loss related to meaningful clinical improvement.[5] Given the limited number of pharmacologic agents available to achieve weight loss, there has been an interest in surgical interventions, especially endoscopic modalities. With the association between NAFLD, morbid obesity, and metabolic syndrome, studies have suggested a role

for bariatric surgery. In morbidly obese patients with NAFLD, bariatric surgery has been shown to reduce inflammation, resolve steatosis, and reverse steatohepatitis.[6]

The first sign of NAFLD is accumulation of hepatic fat (steatosis) that progress to non-alcoholic steatohepatitis (NASH), liver inflammation, fibrosis, cirrhosis and hepatocellular carcinoma.[7]

Current NAFLD and NASH treatments include weight loss obtained by life style modification and pharmaceuticals but have poor effects in patients with body mass index >35 kg/m.[8]Some evidences showed bariatric surgery (BS) could alter the course of disease.Now a days weight loss surgery considered unique method to obtain durable and permanent weight loss in morbid obesity obese could reverse metabolic syndrome consequences as well as fatty liver disease, improve liver histopathology and improve insulin resistance (IR).[9]

Predictors of resolution of NAFLD post-surgery are unclear. We evaluated changes in serum ALT levels, an acceptable surrogate when liver biopsy is not feasible, in 219 Teen-LABS subjects 3 years post gastric bypass or vertical sleeve gastrectomy. Three years post-surgery, median BMI declined to 38.4 from 52.6 kg/m^2 at baseline ($p<0.01$). Median ALT (IU/L) decreased between baseline (36.5 ± 2.6 [95% CI: 31.4, 41.7]) and 6 months after surgery (28.9 ± 2.5 [95% CI: 24.4, 33.9]), and remained stable at 1, 2 and 3 years, all $p<0.01$ vs. baseline. 58% had abnormal ALT at baseline, which decreased to 10% at 3

years. After adjustment by logistic regression, 6 month improvement in BMI, fasting glucose, HOMA-IR, triglycerides, TG/HDL, and HDL were independently associated with reduced ALT. Improvement in metabolic indices remained significantly associated with decline in ALT, after adjusting for BMI

Based of new statements of IFSO (International Federa- tion for the Surgery of Obesity and Metabolic Disorders), bariatric surgery leads to improvement and resolution of NAFLD and NASH in morbid obese patients.[10]Recent studies demonstrated that bariatric surgery; result in improvement of biochemical and histologic parameters of liver, such as non-alcoholic steatosis and steatohepatitis, liver fibrosis, lobular inflammations, chronic portal inflammations and even hepatocellular carcinoma.[11]

Because obesity, diabetes mellitus and hyperlipidemia (HLP) are among the risk factors for the NAFLD progression, so, weight loss induced surgery, improves these comorbidities together with NAFLD.[12]

Actually, the indications of surgery in NAFLD/NASH are the same as in morbid obesity and are BMI equal or more than 40, or 35 < BMI < 40 with any evidence of metabolic syndrome or related comorbidities.

Also bariatric surgery is indicated in overweight patients with medical-resistant severe metabolic complications.[13]

The mechanism of how bariatric surgery plays a role as a potential treatment for NAFLD is also complex and not fully understood. Bariatric surgery is likely to have potential benefits in ameliorating the factors such as insulin resistance, lipid profile, inflammation, weight loss, and adipokines that contribute in a marked way to the pathogenesis of NAFLD.[14]

There are 3 principles of bariatric operations, categorized in respect to their mechanism: (1) Restrictive procedures, which decrease the stomach size to limit the intake of solids; (2) malabsorptive procedures, which limit the absorption of nutrients by shortening the small intestine; thus decreasing the surface area that is exposed to food; and (3) combined, restrictive, and malabsorptive. Laparoscopic adjustable gastric banding (LAGB), sleeve gastrectomy (SG), and gastric bypass (GBP) have become the most preferred procedures worldwide. Restrictive and combined procedures have shown very promising effects on liver function and histology.[15]

FibroScan is an emerging tool for noninvasive assessment of NAFLD. The advantages of TE include assessment of an area a hundred times bigger than a biopsy sample, and good correlation with hepatic steatosis and fibrosis. Previous studies have reported that FibroScan correlates with fibrosis and hepatic steatosis in patients with Hepatitis B virus, hepatitis C virus, and NAFLD with BMI of30 kg/m^2.[16]

After bariatric surgery, there was a significant decline in median ALT (P o .001) and AST (P o .001) levels. Stratopoulas et al. and Dixon et al. reported a significant decline in mean ALT and γ –GlutamylTrans-peptidase at 18 and 26 months' follow-up after surgery.[17]

Bariatric surgery in obese patients with NAFLD results in sustained and significant weight loss and improves NASH, according to the current literature. SG is considered a safe and effective option for these patients, as long as appropriate long-term follow-up is provided. Existing data are not sufficient to recommend general use of this surgical procedure in patients that exhibit NAFLD. The burden of obesity associated comorbidity such as NAFLD in selected patients may prove risky and future studies and a long-term risk analysis of patientswith obesity associated with liver disease.[18]

6.3 AIMS AND OBJECTIVES OF THE STUDY

To evaluate the effects of bariatric surgery on non alcoholic fatty liver disease in morbidly obese patients.

7. MATERIALS AND METHODS

This prospective study will be conducted at Dayanand Medical College and Hospital, Ludhiana and include patients admitted for morbid obesity with nonalcoholic fatty liver disease.

In this prospective study, all consecutive patients undergoing bariatric surgery (between November 2018 to February 2020) as per standard National Institute of Health guidelines will be included; that is, patients with body mass index (BMI)>35 kg/m2 or patients with BMI>32 kg/m2 and associated co-morbidities, such as type 2 diabetes, hypertension, obstructive sleep apnea, and gastroesophageal reflux disease.

•Patients will be analysed on the basis of pre-op and post-op FIBROSCAN and NAFLD fibrosis score, fibroscan will be done at 6 months of interval of the surgery and nafld fibrosis score will be conducted at 3 months and 6 months of time period

•The nafld fibrosis score is validated, noninvasive tool for identifying patients whose NAFLD has advanced to liver fibrosis. It relies on readily available clinical information and routinely measured laboratory data. It is assessed by laboratory values (serum glucose, platelet count, albumin, AST/ALT ratio) and readily available patient characteristics (age, BMI, and diabetes status)

7.1 SOURCE OF DATA

Patients coming in Department of Surgery of Dayanand Medical College and Hospital with morbid obesity.

7.2 METHOD OF COLLECTION OF DATA

Patients coming in Department of Surgery of Dayanand Medical College and Hospital with morbid obesity will be taken up for the study. A group of morbidly obese patients aged 45 ± 5 years and with a body mass index > 35 kg/m2 who underwent sleeve gastrectomy and mini gastric bypass operations had paired fibroscan with nafld fibrosis score, the first at surgery and the second after 6±3 mo of weight loss. Fibroscan and nafld fibrosis score will be assessed and compared. The presence of metabolic syndrome, anthropometric and biochemical variables will also assessed at baseline and at the time of second evaluation.

INCLUSION CRITERIA

Patients with body mass index (BMI)>35 kg/m2 or patients with BMI>32 kg/m2 and associated co-morbidities, such as type 2 diabetes, hypertension, obstructive sleep apnea, and gastroesophageal reflux disease.

EXCLUSION CRITERIA

1) patients undergoing revision surgery, (2) patients with alcohol consumption>28 units/week for men and>18 units per week for women, (3) patients with hepatitis B or hepatitis C infection, (4) patients on drugs known to induce fatty liver or insulin sensitization such as estrogens, tamoxifen, amiodarone, methotrexate, and glitazones.

NUMBER OF CASES

Patients admitted with Obove mentioned inclusion criteria and nonalcoholic fatty liver disease in a 1.5 year of time period extending from January 2019 to June 2020

STATISTICAL ANALYSIS

Data will be described in terms of range; mean ±standard deviation (± SD), median, frequencies (number of cases) and relative frequencies (percentages) as appropriate. Comparison of quantitative variables between the variables will be done using paired Student t-test. A probability value (*p* value)less than 0.05 is considered statistically significant.All statistical calculations will be done using SPSS (Statistical Package for the Social Science) SPSS 21 version statistical program for Microsoft Windows

7.3 DOES THE STUDY REQUIRE ANY INVESTIGATIONS OR INTERVENTIONS TO BE CONDUCTED ON PATIENTS OR OTHER HUMANS OR ANIMALS? IF SO, PLEASE DESCRIBE BRIEFLY.

Yes, patients presenting included in the study will have to undergo hematological, biochemical, radiological investigations. For this patients included in the study will be informed regarding their inclusion and written informed consent will be taken for all the patients included in the study.

7.4 HAS ETHICAL CLEARANCE BEEN OBTAINED FROM YOURINSTITUTION IN CASE OF 7.3?

Attached herewith

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PROFORMA

Date: S.No:

Name: OPD/WARD:

Age: Sex:

CR No.: DOA: DOD:

Address:

History of present illness:

Past history:

Medical treatment for obesity

Any other therapy for obesity

- **Diet therapy duration(Kcal/day)**
- **Physical Therapy**

Signature of Candidate **Signature of Supervisor**

Other comorbidities-

Hypertension

CAD

History of DM

Previous Treatment/surgery/intervention biopsy:

Occupation:

General Physical examination:

BP-	Temp-	SpO2 (on room air)-
PR-	RR-	
Pallor-	Cyanosis-	Lymphadenopathy-
Clubbing-	Edema-	Icterus-

Petechie/Purpura-

Others:

_____ _____

Signature of Candidate Signature of Supervisor

Significant Systemic Examination:

Chest-

Per-Abdomen-

CVS

CNS

Investigations:

Any Other Investigations

Signature of Candidate

Signature of Supervisor

	PRE-OPERATIVELY	AFTER 3 MONTHS	AFTER 6 MONTHS
Blood Pressure (Systolic/diastolic) (mm Hg)			
Height(m^2)			
Weight(Kg)			
BMI(Kg/m^2)			
Hip/Waist ratio			
S.TRIGLYCERIDES			
LDL			
HDL			
HbA1C			
Fasting Glucose			
SGOT			
SGPT			
PLATELETS (X109/L)			
ALBUMIN			
FIBROSCAN CAP score Fibrosis score		XXX	
ANTI-HCV		XXX	XXX
Hbs Ag		XXX	XXX
USG ABDOMEN		XXX	

Surgery performed:

Signature of Candidate

Signature of Supervisor

INFORMED PATIENT CONSENT

Date: _____

I.....D/o, W/o,

S/o..... .....am willing to participate in the study **"A PROSPECTIVE STUDY TO SEE THE IMPACT OF BARIATRIC SURGERY ON NON ALCOHOLIC FATTY LIVER DISEASE (NAFLD) IN MORBIDLY OBESE PATIENT"** The procedure with its complications has been explained to me in local vernacular language. I have fully understood that no special tests and extra expenditure in any form, merely for the purpose of thesis is being carried out. I am free to opt out the study any time without assigning any reason.

....

Signature of the Witness Signature of the patient

Name:

Signature of Candidate **Signature of Supervisor**

मरीज अनुमति फार्म

तिथि _____

मैं _____ पत्नी/पुत्र/पुत्री _____ निवासी _____ इस शोधकार्य **"A PROSPECTIVE STUDY TO SEE THE IMPACT OF BARIATRIC SURGERY ON NON ALCOHOLIC FATTY LIVER DISEASE (NAFLD) IN MORBIDLY OBESE PATIENT"** में भाग लेने के लिए सहमत हूँ। मैं इस शोधकार्य में भाग लेने के लिए अपनी स्वेच्छा से तैयार हूँ। मैं पुष्टि करता/करती हूँ की यह स्वीकृति देने के लिए मुझे कोई आर्थिक प्रलोभन नहीं दिया गया और न ही दबाव डाला गया है। मुझे इस शोधकार्य का उद्देश्य स्पष्ट कर दिया गया है और मुझे यह विश्वास है की यह विज्ञान और मानवता के लाभ के लिए है। मुझे इस शोधकार्य में होने वाले जोखिम के बारे में समझा दिया गया है और मेरी समझ में यह जोखिम बहुत मामुली है।

मैं इस शोधकार्य में भाग लेने के सम्बन्ध में किसी भी समय अपनी अस्वीकृति व्यक्त कर सकता/सकती हूँ और मेरे अस्वीकृति व्यक्त करने का मेरे इलाज पर कोई प्रभाव नहीं पड़ेगा।

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गवाह के हस्ताक्षर
नाम:

_____
रोगी के हस्ताक्षर/अंगूठे का निशान
नाम:

_____
अन्वेषक के हस्ताक्षर

_____
विद्यार्थी के हस्ताक्षर

ਮਰੀਜ਼ ਰਜ਼ਾਮੰਦੀ ਫਾਰਮ

ਮਿਤੀ_____

ਮੈ_____ਪਤਨੀ/ਪੁੱਤਰ/ਪੁੱਤਰੀ_____

ਵਾਸੀ _____

"A PROSPECTIVE STUDY TO SEE THE IMPACT OF BARIATRIC SURGERY ON NON ALCOHOLIC FATTY LIVER DISEASE (NAFLD) IN MORBIDLY OBESE PATIENT" ਤੇ ਚਲ ਰਹੀ ਖੋਜ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਰਜ਼ਾਮੰਦ ਹਾਂ। ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਮੈ ਆਪਣੀ ਮਰਜ਼ੀ ਨਾਲ ਤਿਆਰ ਹਾਂ। ਮੈ ਇਹ ਦਸਣਾ ਚਾਹੁੰਦਾ ਹਾਂ/ ਚਾਹੁੰਦੀ ਹਾਂ ਕੇ ਇਹ ਰਜ਼ਾਮੰਦੀ ਦੇਣ ਵਾਸਤੇ ਮੈਨੂ ਕਿਸੇ ਵੀ ਤਰਾਂ ਦਾ ਲੋਭ-ਲਾਲਚ ਨਹੀ ਦਿੱਤਾ ਗਿਆ ਅਤੇ ਨਾ ਹੀ ਕੋਈ ਦਬਾ ਪਾਇਆ ਗਿਆ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਦਾ ਮਨੋਰਥ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਭਰੋਸਾ ਦਵਾਇਆ ਗਿਆ ਹੈ ਕੇ ਇਹ ਖੋਜ ਵਿਗਿਆਨ ਅਤੇ ਮਾਨਵਤਾ ਦੀ ਭਲਾਈ ਲਈ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਵਿਚ ਹੋਣ ਵਾਲੇ ਖਤਰੇ ਬਾਰੇ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਯਕੀਨ ਹੈ ਕੇ ਇਹ ਖਤਰਾ ਬਹੁਤ ਮਾਮੂਲੀ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਦਾ ਢੰਗ – ਤਰੀਕਾ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ।
ਮੈ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਬਾਰੇ ਆਪਣੀ ਰਜ਼ਾਮੰਦੀ ਕਿਸੇ ਵੀ ਸਮੇ ਵਾਪਸ ਲੈ ਸਕਦਾ ਹਾਂ/ ਸਕਦੀ ਹਾਂ ਅਤੇ ਮੇਰੀ ਰਜ਼ਾਮੰਦੀ ਵਾਪਸ ਲੈਣ ਦਾ ਮੇਰੇ ਇਲਾਜ ਤੇ ਕੋਈ ਅਸਰ ਨਹੀ ਹੋਵੇਗਾ।

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ਗਵਾਹ ਦੇ ਹਸਤਾਖਰ ਮਰੀਜ਼ ਦੇ ਹਸਤਾਖਰ

ਮਿਤੀ_____ ਮਿਤੀ_____

ਪਤਾ _____ ਪਤਾ _____

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ਨਿਰੀਖਕ ਦੇ ਹਸਤਾਖਰ

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ਸਿਖਿਆਰਥੀ ਦੇ ਹਸਤਾਖਰ